New Orleans Medical
and
Surgical Journal

$4.00 Per Annum, 35c Per Copy
Vol. 103—No. 6

DECEMBER, 1950

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

VARIOUS PEDIATRIC ENDOCRINE PROBLEMS

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There are a number of recognizable, important and treatable endocrine problems in children which practitioners encounter. Also there are conditions called endocrinal which are not and these should be so recognized.

NONENDOCRINE PROBLEMS

Obesity. Most cases of obesity are due mainly to the habit of overeating. However, very likely some are mainly of endocrine origin. Practitioners of long experience have seen children acquire excessive or not so excessive appetites and become obese; for instance, after basal skull fractures, encephalitis and hydrocephalus. Hypothalamic lesions linked up to pituitary lesions often are the cause. Some of these children are dwarfed and slow to mature; some even develop diabetes insipidus—all possibly due to failure of the pituitary to develop these respective hormones to be handled properly in the hypothalamus.

Pituitary extracts on the market are ineffective in controlling excess appetite or weight. Response to thyroid extract is not full proof of a primary or even secondary thyroid or thyrotropic pituitary lack; thyroid here has an effect of raising the basal rate and burning excess tissue—a calorigenic effect. The best therapy for obesity is development of the will to cut down on excess food intake. The physician must gain the confidence and cooperation of the child on a low calorie diet. Behavior and psychological problems must be overcome. Anorexigenic drugs, like dexedrine, are justifiable and recommended.

Mongolism. This developmental defect is not due to endocrine disturbance; definitely it is not due to thyroid lack. However, individuals with mongolism are entitled to develop thyroid lack in the same frequency as other children. The following findings, which are only a few characteristic of mongolism, are not found or rarely found in hypothyroidism: the slant eyes with epicanthic folds, the lax muscles and joints, the liveliness, the congenital heart lesions, and failure to respond satisfactorily to thyroid extract.

Thyroid extract can be given to these patients if for no other reason than to convince predetermined parents that this is not hypothyroidism and that thyroid extract will do no more than give their child a slight lift, proving thereby that this deficiency is not of thyroid origin. Do not hesitate to tell a parent, if this is thus proven to you, that their child with mongolism is not thyroid deficient but was born with primarily a generalized developmental defect involving mainly the brain. There is evidence that maternal disease in the first trimester may cause this.

Presented at the Seventieth Annual Meeting of the Louisiana State Medical Society, April 26, 1950, in Baton Rouge.

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Journal Series No. 905, University of Arkansas.

Figure 1. Mongolism not due to thyroid or other endocrine deficiency.

Figure 2. The boy on the left is tall, obese, and has normal genitalia—the Neyroth-Cushing type. The girl in middle is short and not matured—the Levi-Lorain type.

HYPOPITUITARISM

A number of entities in childhood have been ascribed to hypopituitarism, among them obesity, which has never been causative. However, among those definitely or strongly suspected as being caused by hypopituitarism are diabetes insipidus, and the syndromes of Fröhlich, Levi-Lorain and Neuroth-Cushing. (Figure 2.) Let us consider the last three. The term "Fröhlich syndrome" should be reserved for the definite entity he described, and which syndrome occasionally is remedied by surgery. In 1901, Fröhlich described a dwarfed, obese, genitally retarded 14 year old whose hypopituitarism, of at least growth and gonodal hormones (if not others, then unrecognizable), was due to craniopharyngioma (the usual cause). He was greatly relieved by drainage and partial removal of a cyst. The true syndrome is not common. The term Fröhlich should not be used so freely as it has been of late years for every fat adolescent with slight retardation of genital development (more pseudo than real), for if the apron of mons fat is retarded, the boy's genitalia often appear quite normal, and he has normal or better than normal height.

The Neuroth-Cushing syndrome, described by both writers independently about 1912, is the most common hypopituitarism encountered, if it that at all. These individuals may lack a bit of pituitary gonadotropin and whatever hormone or mechanism that controls appetite. Their growth hormone production and utilization, however, are usually better than normal; their epiphyseal age is usually accelerated slightly. This syndrome may well be a normal physiological variant, for over two-thirds of them normalize after puberty, which in girls often is at an earlier age than the average. Weight control is the main therapy. Gonadotropins may be indicated for genital retardation just before, during, or just after the average age of puberty. In boys, anatomical genital defects (hernia, hydrocele, and immobile ascended testis), should be surgically corrected (hernia and hydrocele by the age of 2 years, and immobile perpetually ascended testis by 4 to 8 years, mobile inconstantly ascended testis right after the average age of puberty—12 to 14 years).

The third syndrome recognizable clinically is that of Levi-Lorain; it is not common. It is characterized by dwarfism and genital infantilism, but no obesity, as contrasted with Fröhlich's. Occasionally other pituitary deficiencies may occur in this; diabetes insipidus and tumor or cyst may be present. Mentality as a rule is unimpaired; in Fröhlich's, often these fat children are like "Peck's bad boy"—they have good mentality but lack concentration and some degree of memory, and are too playful and inattentive in school. The Levi-Lorain patients lack pituitary growth and gonadotropin hormone. In addition to their dwarfism, the lack of secondary sex characteristics is so marked as to be a great psychological hurdle. Gonadotropins are distinctly indicated here; the pituitary ones are theoretically indicated. There are none on the market, so chorionic gonadotropin from human pregnancy urine is used as it has an action similar to the pituitary one. Gonadal hormones, testosterone or estrogen, also may be helpful and can be used along with gonadotropins. Pituitary growth hormone to date has not been helpful in humans; the newest one of Evans and Li is toxic. Testosterone and estrogens are now better growth stimulants.

HYPOTHYROIDISM

This is not too commonly encountered. It is very important to recognize this deficiency early in its career in order that the treated child will acquire the maximum mental development. The physical improvement is only of secondary importance. (Figure 3.)

Figure 3. Marked thyroid deficiency; rapid therapeutic response.

Cretinism. In those areas of the world where cretinism is endemic, it is characterized by goitre, mental retardation, familial recurrence, and failure to respond very much to thyroid extract. This form of hypothyroidism is rare in this country. However, congenital failure of the gland to be present, or presence of the gland very deficient in production of a normal amount of thyroid hormone, may be more prevalent than realized heretofore. Examining the child's photographs from birth to two years often reveals such an early onset. The family may not notice the onset until younger siblings start to outgrow the patient when the latter is 4 or 5 years old, or until the patient first goes to school and does not do well intellectually. I have encountered this several times in recent years; such failure to have the patient treated early is often due to lack of parental observation. (Figure 4.)

Figure 4. The five year old boy was noted to have his three year old brother outgrow him; however, he was retarded at twenty-one months.

Acquired hypothyroidism probably is still the more common occurrence. As a rule, these patients are not so deficient as the congenital ones. Temporary hypothyroidism occurs but it is not common, not marked, and therefore, not easily recognized. A 9 year old girl during six or eight months developed vitiligo, became slightly overweight, then failed to do well at school for she even became sleepy. Her hair dried slightly and slight myxedema appeared. The basal metabolic rates were very low. She responded to thyroid, relapsed when it was stopped, responded again, and after six months of such therapy did not relapse when thyroid was finally stopped. Her recovery was complete.

She did not have marked deficiency long

enough to cause dwarfism. Note that all of the findings did not develop at the same time and to equally comparable degrees; though basal metabolic rate was very low, the myxedema of the face, for instance, was only slight, and there was none detectable elsewhere. The genesis of the findings in all forms of thyroid deficiency is slow, the findings are unequally developed and some tissues can do normally for years with smaller amounts of thyroid hormone than others before finally requiring excess amounts to maintain such normality. This response of target organs and tissues is all important in understanding the picture of hypothyroidism. This concept, also, holds true for practically all syndromes and diseases.

One should not expect to detect all of the classical findings described in our textbooks, especially those published some years ago. Dwarfism takes years to develop; myxedema may be hidden from sight, as in the larynx or abdomen or in the knee joints, and aften is mild in degree. Dry, coarse, thick skin and hair may be localized and not generalized. The outer thirds of the eyebrows may still be present. Many other trivia could be detailed to point out the lesson that all hypothyroids do not have all of the classical findings described for many years in our textbooks.

A basal metabolic rate is not too reliable unless it is repeatedly and markedly low. A blood cholesterol should be definitely and not equivocally high. To be reliable, epiphyseal age by x-ray should be retarded more than six months in infants and more than one and one-half years in children over 2 or 3 years old. The writer is no exception to the usual tendency of investigators to talk up his work; so the polite reader is asked to bear with me on the usefulness of radioactive iodine as a laboratory aid. A harmless test dose—5 to 10 microcuries of I^{131}—is given by mouth. The thyroid takes up normally 10 to 25 per cent and the kidney excretes 45 to 65 per cent in forty-eight hours. The cretin takes up practically none and excretes 70 to 90 per cent; the partial hypothyroid takes up about 5 to 10 per cent and excretes about 65 to 80 per cent. The excretion may be more reliable as a test of thyroid function. The test possibly may prove so practical for children in equipped laboratories as to displace the difficult and somewhat unreliable basal metabolic rate.

Early treatment is desirable principally to insure the maximum mental development. There is less difficulty now in obtaining a potent thyroid extract; it should be in a dark bottle and not too old. The daily doses for maintenance usually are: 2 to 4 months about 6 mgms.; from 4 to 8 months about 12 mgms.; 8 to 12 months about 18 mgms.; 12 to 24 months about 25 to 50 mgms.; 2 to 4 years about 30 to 100 mgms.; 4 to 12 years about 60 to 200 mgms. This should be started at a lower dose. The total daily ration can be given at one time; the drug is cumulative. Raise the dose every seven to ten days until toxicity occurs; then maintain on a subtoxic dose. Do not let the parents worry about questionable toxic manifestations. The drug being cumulative, can be stopped for four to seven days every four to six weeks, if necessary, to control toxicity but principally to continue therapy for life, which will be necessary.

It is surprising how well the mentality will develop in some of even congenitally deficient thyroid patients. If treatment is begun early, 25 per cent can become normal mentally and 50 per cent will be subnormal. The most usually accomplished is the prevention of further deterioration.

HYPERTHYROIDISM

While uncommon in children in the South, this is remedial without serious sequelae in growth or development. There are two effects noted in serious cases of long duration: gigantism, as the result of the great metabolic stimulation, and failure to mature properly at the expected age due to the natural antagonism of thyroid hormone and estrogen. (Figure 5). The mysterious cause is still being sought. Some now feel it is because of excessive thyrotropic hormone. All goitre is not due to lack of iodine intake; there are certain drugs and foods which interfere with proper utilization of iodine and cause goitre. Examples are:

Figure 5. Severe hyperthyroidism.

thioureas, thiothiazolines, thiooxazolines, aminothiazole, aminobenzenes, thiocyanate, turnip, rutabaga, cabbage, Brussels sprouts, cauliflower, and such *Brassica* vegetables and their seeds.

For the mildly toxic cases medical therapy is still successful. This consists of Lugol's solution or other forms of iodine, extra rest usually bed, high caloric diet, and perhaps sedation. If such a mild case, after arrest, should relapse (and this is the natural history of this disease) seriously then one should think of roentgen therapy or even surgery.

For severely toxic cases, surgery offers the best results in the long run. This should be done in one stage, during a remission, leaving only 4 to 6 grams of gland (it will regrow; the cause of growth is not removed by operation). Done by an experienced surgeon, the mortality is 0.5 per cent or less. Deficiency postoperatively is only temporary and not serious. Growth, development and maturity are unimpaired. Roentgen therapy only if well done, is very helpful and can be tried if medical management fails in severe cases but I think surgery, if well done, is better. There is nothing that I^{131} therapy does, which roentgen therapy cannot do. However, I^{131} therapy has some advantages—ease of oral administration, lack of radiation sickness, relative minimum or radiation effects elsewhere in the body, low cost and in the future more definite safety. Here are some of our results. (Table 1).

These children had the disease for six months to two years before and have re-

TABLE 1

HYPERTHYROIDISM BEGINNING IN CHILDHOOD—SUCCESSFULLY TREATED WITH I-131

	Name	Age	Sex	Estimated Thyroid Weight		Basal Metabolic Rates		Protein-Bound Iodine, Mg. Per 100 cc.		Total Dose I-131, Microcuries	Uptake I-131 In Thyroids	Time from Initial Dose of I-131 to Return to Normal
				Before	After	Before	After	Before	After			
				Gm.	Gm.	%	%					Months
1.	E. F.	17	F	15 (post-operative recurrence	5 (barely palpable)	+16	−3	7.2	5.4	800		2½
2.	G. M.	9	F	20-25	Less than 10+	+22		17.4(?)	5.2	2000	1462	3
3.	E. M.	17	F	35	Less than 35	+26	−5	11.4	5.6	4500	2325	5
4.	E. J. N.	9	F	25	12+	+2	−25	8.4	5.0	3000	600	5
5.	B. W.	16	F	35-40	25-30	+27	−13	11.9	5.2	4000	1840	4

mained "cured" since receiving I^{131}, which was several years ago.

Propylthiourea, the choice of the thioureas, is helpful, must be used for weeks or months, is indicated for mild cases or preoperatively, and must be watched for the 2 per cent who will become toxic. Though it arrests toxicity, 35 to 50 per cent will relapse.

ADRENAL CORTICAL LESIONS

Insufficiency. Increasingly in the past few years a syndrome has been described in newborns characterized by the concomitant occurrence of lack of the salt and water controlling hormone (a partial Addisonism) and excess of the adrenal cortical androgen. At about two weeks of age they suffer from vomiting, occasionally projectile, loose frequent bowel motions, great dehydration and weight loss, shock and low blood pressure, all evidence of lack of salt and water hormone. Some have been operated upon as instances of pyloric stenosis; none has been found and often death ensued. Large fetal adrenals were found with overgrowth of the androgenic zone (inner fetal zones) with overproduction of adrenal androgen accounting for precocious masculinization, which often develops in those who survive. Laboratory tests show, among other findings in the blood, low sodium and chloride and CO_2, high potassium, NPN, R.B.C., and in the urine low sodium and high chloride and 17-ketosteroids. This syndrome has to be kept in mind to be diagnosed. It accounts for a number of unexplained deaths in the past. The wonder is, what causes it. So far, no disturbance during the mother's pregnancy has been suspected.

It is controlled by the following: Sometimes only by saline infusions or sodium chloride orally, but more often by saline plus desoxycorticosterone acetate injections; if that salt-water hormone controls, then subcutaneous pellets of the same are indicated. Occasionally the condition is only temporary or is manifested only by the salt-water hormone lack. Usually the masculinization does not manifest itself until some months of age; there does not seem to be any control or cure for that.

Excess Androgen Production. Most of the gonadal hormone production of the cortex is androgenic so that masculinization predominates. For some unexplained reason the disturbance occurs about four times more often in individuals primarily female so that pseudohermaphroditism is common. Hyperplasia is about 5 to six times more common than tumor. (Table 2).

Newborns, with also the previously described insufficiency, having had the disturbance in utero are one group due to hyperplasia. Hyperplasia or tumor starting after birth are probably more common than those starting in utero; usually as with tumors starting after birth there is no associated deficiency of salt-water hormone except after surgical removal of cortical tissue.

Tumors or hyperplasia in a girl cause growth of the clitoris which is almost pathognomonic and almost invariably present. Masculinization also needs no full explanation and its occurrence at an early age in

TABLE 2

		Tumor or Hyperplasia	Other Conditions Causing Similar Syndromes
In intrauterine	Pseudohermaphroditism		
In infancy and childhood	Boy	Sexual precocity	Pineal tumor. Testicular tumor. Cerebral tumor.
	Girl	Pseudosexual precocity with pubic hair. Enlarged clitoris.	True sexual precocity—physiological. Adult genitalia. Early menstruation. Granulosa cell tumor of the ovary.

this disturbance is not unusual. The differentiation between tumor and hyperplasia can be solved partly by: Cushing's disease (different from the one described under pituitary disturbances) is usually due to adrenal cortical tumor and the masculinization is accompanied by hypertension, insulin-resistant diabetes, obesity, plethora, and blue abdominal striae. Cushing's is thought to be due to a combination of excess production of androgen and 11-oxycorticoids (carbohydrate-regulating hormones of "S" hormones) of the cortex. Tumor sometimes can be felt or visualized in x-ray by calcification (due to old hemorrhage) or pyelographic demonstration of a distorted downwardly displaced kidney pelvis or by air injection about the adrenal which roughly reveals its size. The differentiation between tumor and hyperplasia and their lateralization is difficult at times preoperatively. It is thought that excess urinary excretion of beta 17-ketosteroids is diagnostic of tumor, and excess of the alpha fraction is diagnostic of hyperplasia. Operative exploration of both sides is often indicated not only for the localization but also for the type of pathology, and what is most important the surety that the partner gland is present before removal of the abnormal gland. Saline, whole adrenal cortical extract and desoxycorticosterone acetate should be available during the removal of the abnormal gland for the remaining partner may have been depressed for a long time before and may require several days to take over full needs of the body for these vital cortical hormones.

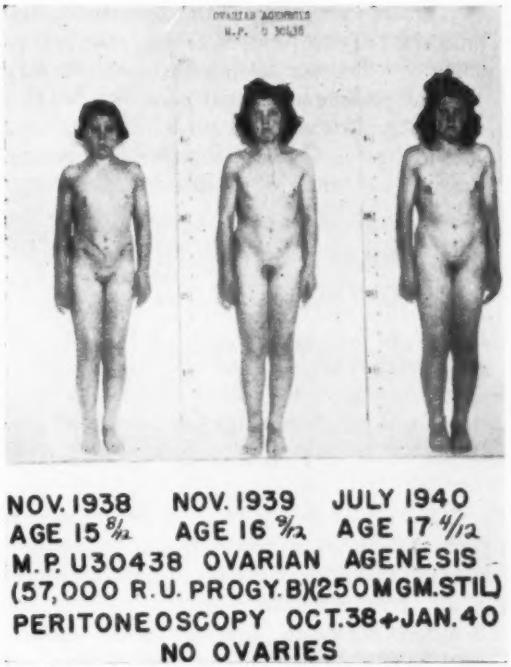

Figure 6. Ovarian agenesis responds well to estrogens.

OVARIAN AGENESIS

While not common, this deficiency is remedial by oral estrogen therapy. It is due to congenital failure of the primordial ovarian follicles to mature though there is an excess of pituitary gonadotropin. (Figure 6).

Such girls are short for their age, most being around 52 inches; they have a maturation status of about a year old and do not menstruate though they may be 14 or 16 years or older; they have a childish facies, often a web-neck, increased carrying angle at the elbows and congenital heart lesions. Epiphyseal development is slightly retarded and there is excess of urinary pituitary gonadotropin, but estrogen is practically nil. The clinical differentiation from pituitary dwarfism and infantilism (the Levi-Lorain syndrome) is difficult; the hormone pattern described above is very helpful.

Treatment with estrogens orally is very helpful though not curative. The secondary sex characteristics develop almost to a normal status within about one or one and a half years; cyclic vaginal bleeding can be induced by administering the estrogen for three weeks and then withdrawing it for one week. Natural estrogens, like ethinyl estradiol (Estinyl of Schering) can be given. Estinyl is potent, not toxic, and .1 mgm. daily can be given orally. Synthetic estrogen-like nuterids like stilbestrol can be given orally, 1 to 2 mgms. daily; occasionally, these materials are slightly upsetting but not too toxic. Initial nausea is usually overcome by a rest period before readministration.

The defective primordial follicle does not mature but the endometrium changes cyclically. The corpus luteum does not form so that pregnancy is not possible. Physical reconstruction of such girls relieves them of psychological disadvantages; I have had several who thus were able to marry.

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MATERNAL, FETAL AND INFANT MORTALITY IN LOUISIANA*

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AND

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This paper has a twofold objective: to demonstrate that through the years as a result of the cooperative efforts of physicians, public health workers, nurses, and hospital personnel that maternal and infant mortality is steadily diminishing and to focus attention on problems still confronting us in the field of maternal and infant care in Louisiana.

As you will note by referring to Table 2, the over-all maternal death rate has decreased from 11.1 deaths per 1000 live births in 1925 to 1.2 in 1949. When the maternal death rate is computed for whites and nonwhites you will note that the white rate has decreased from 8.3 in 1925 to 0.7 in 1949 and the nonwhite rate for the same years has decreased from 15.8 to 2.1. It is immediately obvious, however, that the maternal death rate for nonwhite women is three times as great as that of white women for the year 1949. By reference to 1925 we find the nonwhite rate was only 1.8 times as great as the white rate. Therefore, while tremendous progress has been made in reducing both the white and nonwhite rate the white rate has lowered much faster than the nonwhite. If you will refer to Table 4 you will find listed the ten leading causes of death in 1925. Comparison of this list with the ten leading causes of death in 1948 reveals that puerperal toxemias are still the first cause of maternal deaths, but that the maternal death rate for this cause has decreased from 4.1 deaths per 1000 live births in 1925 to 0.3 in 1949. Puerperal septicemia which was the second leading cause of death in 1925 with a rate of 2.0 is in fourth place in 1948 with a rate of 0.2. Puerperal hemorrhage which was in fifth place in 1925 has now moved into third place, but the rate has decreased from 1 to 0.2. Reference to the table also reveals that while all rates are decreasing that certain causes are becoming increasingly prominent as causes of maternal deaths when one compares the leading causes in 1925 with those of 1948. Notably these are: toxemias of pregnancy and ectopic gestation.

While we present data in Table 10 relative to maternal deaths by age group for the years used in this study we regret that at this time we do not have rates by age groups for comparative purposes.

In 1925, physicians attended 55.4 per cent of the women delivered of live babies; by 1949, physicians were attending 84.8 per cent of this group. In 1925, infant deaths were 9.1 per cent and maternal deaths were 1.1 per cent of the total live births as compared to 3.8 per cent infant deaths and 0.2 per cent maternal deaths for the same group in 1949.

In 1925, the over-all neonatal death rate (Table 3) was 46.1 per 1000 live births as compared to 27.3 in 1949. When this is broken down into white and nonwhite we find that the neonatal death rate for the white group has decreased from 39.5 in 1925 to 24.8 in 1949 while the nonwhite rate has decreased from 56.6 to 39.3. In 1925, the nonwhite death rate was 1.4 times as great as the white rate, and in 1949, this proportion is essentially unchanged being 1.5 times as great for the nonwhite group. What is significant, however, is the fact that the nonwhite neonatal death rate in

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*Presented at the Seventieth Annual Meeting of the Louisiana State Medical Society, April 26, 1950, in Baton Rouge.

1949 is just what the white neonatal death rate was in 1925.

The infant death rate (Table 1) per 1000 live births in 1925 was 91.1; in 1949, this rate was 37.7. The white rate decreased from 72.2 in 1925 to 33.3 in 1949; while the nonwhite rate which was 1.7 times as great as the white rate in 1925 decreased from 123.1 for that year to 58.6 in 1949, but it still remains 1.7 times as great as the white rate.

The ten leading causes of infant death for the years studied are listed in Tables 12 through 17. Diarrhea and enteritis which were the leading causes of infant deaths in 1925 with a rate of 19.8 are in sixth place in 1948 with a rate of 1.5, a marked decrease. Premature birth which was in second place in 1925 with a rate of 15.3 is in first place in 1948 with an almost unchanged rate of 13.1. Congenital debility in third place in 1925 with a rate of 12.1 has dropped to ninth place in 1948 with a rate of 0.7. Bronchopneumonia in fourth place in 1925 with a rate of 7.5 has risen to second place in 1948 with a rate of 4.6. Congenital malformations, fifth leading cause of death in 1925 with a rate of 4.0 is in third place in 1948 with a rate of 3.6. The sixth leading cause of infant death in 1925 with a rate of 3.2 is birth injuries; 1949 finds birth injuries in fourth place as a cause of infant deaths with a rate of 3.5. The infant death rate from syphilis is one-sixth in 1948 of what it was in 1925; while whooping cough which was in ninth place in 1925 is not even listed as one of the ten leading causes of death in 1949.

The stillbirth rate (Table 18) rose from 37.6 per 1000 live births in 1920 to a peak of 58.4 in 1928. Since 1928 this rate has been consistently decreasing. In 1948, the stillbirth rate is 25.5. However, comparison of the white with the nonwhite stillbirth rate points up the fact very clearly that this over-all rate can be lowered still further by improving prenatal care for the nonwhite group. In 1921, the first year of this study for which figures are available for the white and the nonwhite group, the stillbirth rate for the white group was 39.1 and the rate for the nonwhite group was 75.3. In 1948, the rate for the nonwhite group is 16.9 as compared to 39.2 for the nonwhite group. You will note that in 1948 the rate for the nonwhite group is what the stillbirth rate for the white group was in 1925 (refer to Table 18).

The vital statistics quoted demonstrate the first stated objective of this paper which is to prove that maternal and infant mortality is diminishing. The factors influencing this progress are not important to us at this time as it is very likely that these factors will continue to operate; suffice it to say that this progress is the resultant of the cooperative efforts of all persons interested and active in promoting better maternal and child care programs and that as this cooperative effort is extended in the behalf of mothers and children so will their welfare be advanced.

The second objective of this paper was to focus attention on problems still confronting us. The first and most obvious fact is that the maternal and infant death rate for the nonwhite group is much higher than it is for the white group. While we do not have the figures at hand, at this time, to prove the point it is our opinion, based on experience, that this is the result of two factors: medical care and hospitalization for delivery and illness for a greater number of individuals among the white than among the nonwhite group.

Other problems pointed up by this study are that:

1. Puerperal toxemias and septicemias are still the leading cause of maternal deaths after twenty-three years.
2. Puerperal hemorrhage, toxemias of pregnancy, and ectopic gestation are still formidable causes of maternal deaths.
3. Premature birth is the leading cause of neonatal and infant deaths and almost no progress has been made over the state as a whole in meeting the challenge of prematurity.
4. Bronchopneumonia is still a leading cause of infant deaths and not much

MATERNAL MORTALITY SHOWN BY RACE
LOUISIANA, 1925 - 1949

RATES = NUMBER OF MATERNAL DEATHS PER 1,000 LIVE BIRTHS.

*1949 FIGURES ARE PROVISIONAL.

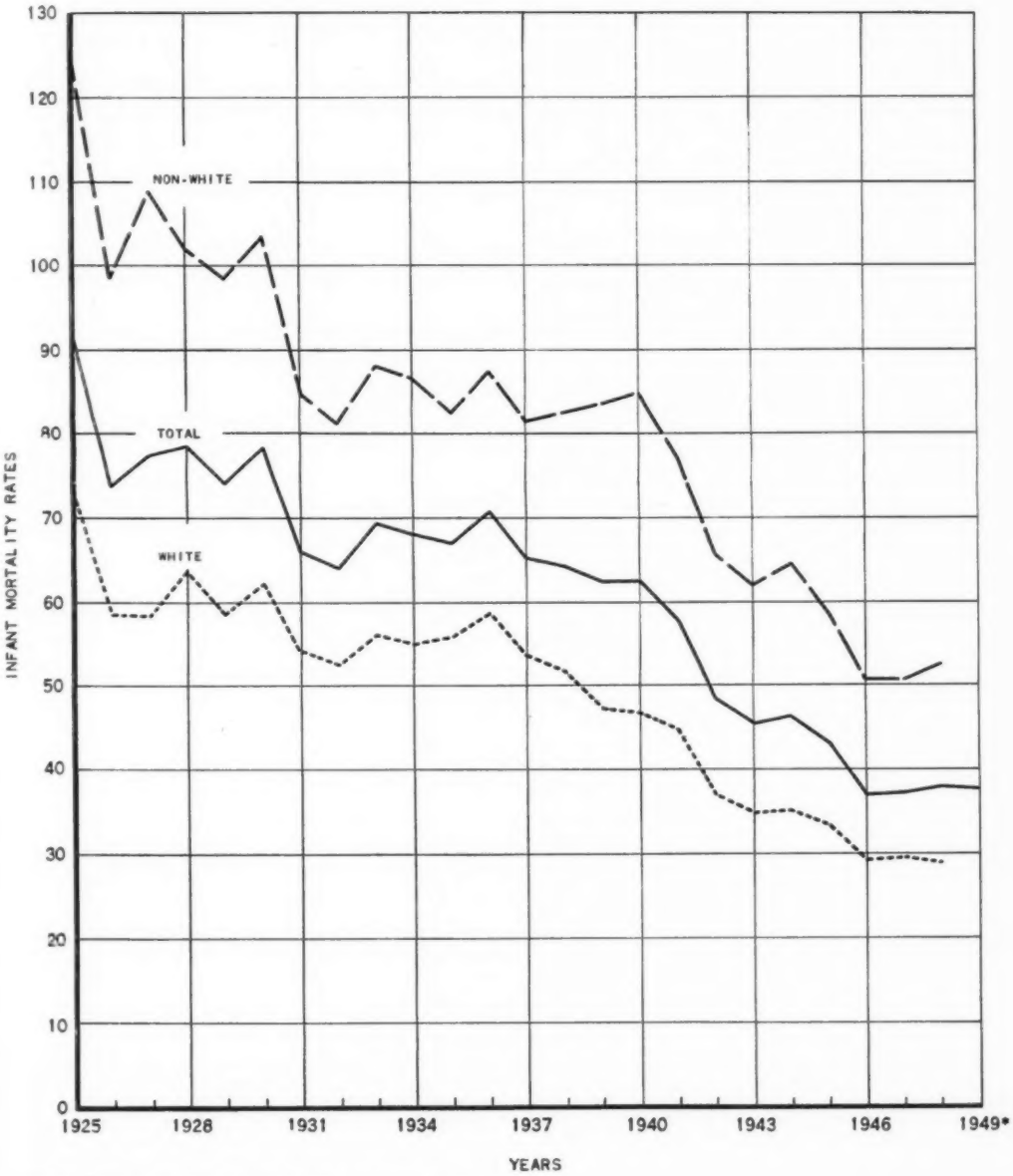
PREPARED BY- TABULATION AND ANALYSIS SECTION

progress has been made in combating it.

5. Congenital malformations and birth injuries are still leading causes of death, and death rates for these conditions remain essentially unchanged from 1925 to 1948.

The question will probably be raised as to why no effort was made to compare Louisiana data with national data. The answer is that while it is nice to know that

INFANT MORTALITY SHOWN BY RACE
LOUISIANA, 1925 - 1949



RATES = NUMBER OF INFANT DEATHS PER 1,000 LIVE BIRTHS.

*1949 FIGURES ARE PROVISIONAL.

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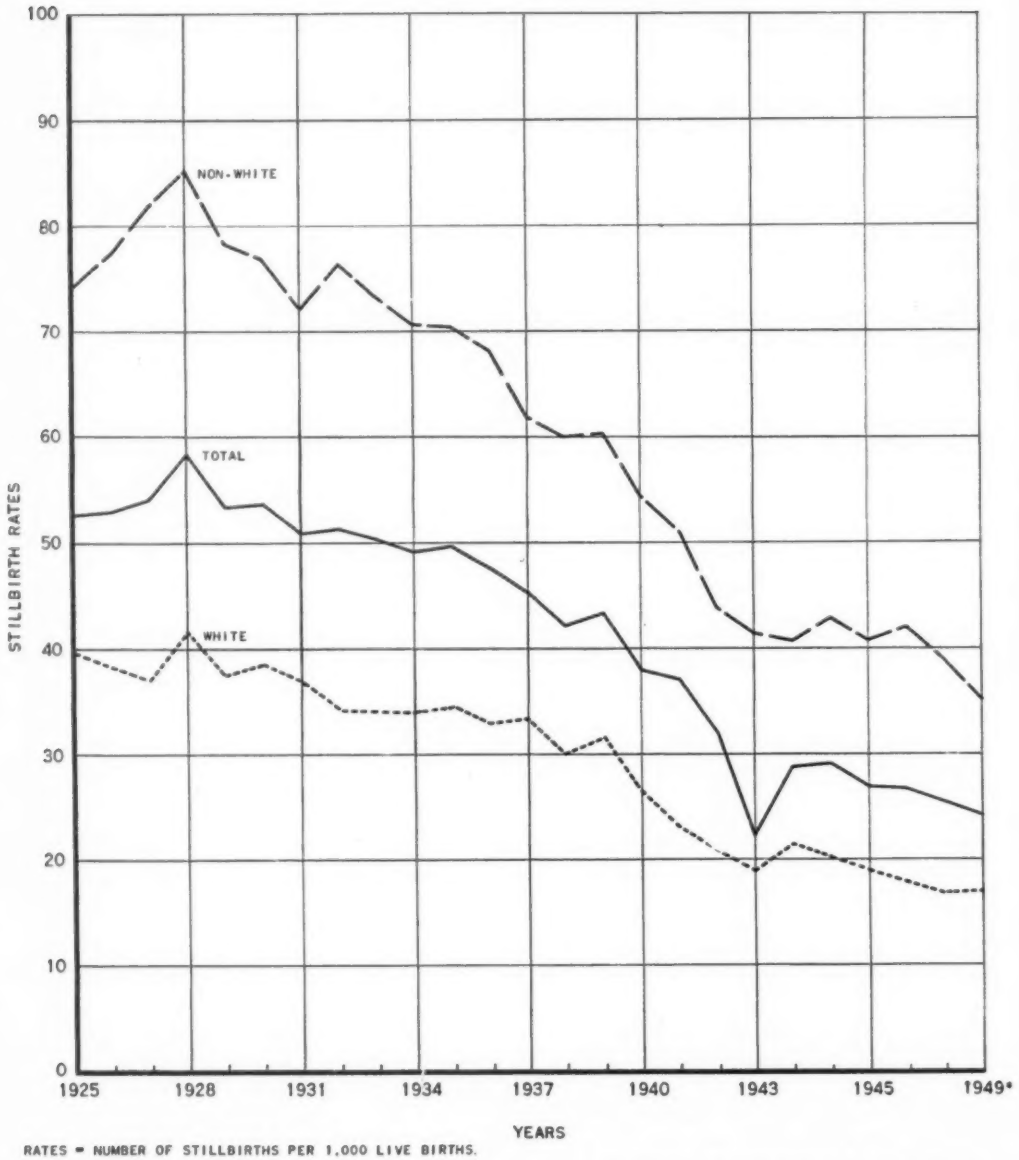
Louisiana compares favorably with the nation as a whole such a comparison is not necessary to show that progress in maternal and infant care is being made in Louisiana, or to point out the particular important problems which confront us at this time.

While the objectives of this paper have

STILLBIRTHS SHOWN BY RACE
LOUISIANA, 1925 - 1949



RATES = NUMBER OF STILLBIRTHS PER 1,000 LIVE BIRTHS.

*1949 FIGURES ARE PROVISIONAL.

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been attained, nevertheless we feel that the following suggestions are in order:

1. A program should be developed to provide hospital delivery service for all expectant mothers.
2. The medical care which is now available to white women should be made available to nonwhite women and an educational program should be developed which would encourage them to seek such medical care.
3. Obstetricians should take a vital interest in the causes of infant deaths as three of the four leading causes of

infant death are the result of factors acting on the baby during pregnancy and at the time of delivery, and before the pediatrician ever sees the baby.

4. A study should be made to correlate maternal and infant deaths with medical care and hospitalization.

TABLE 1

INFANT DEATHS SHOWING NUMBER AND RATES
LOUISIANA, 1925, 1930, 1935, 1940, 1945, 1949

Year	Total	Rate	White	Rate	Nonwhite	Rate
1925	3816	91.1	1902	72.2	1914	123.1
1930	3356	78.3	1623	62.2	1733	103.3
1935	2829	67.0	1367	55.9	1462	82.3
1940	3187	62.5	1399	46.8	1788	84.8
1945	2507	43.2	1183	33.3	1324	58.6
1949	2740	37.7	.....	.....	.....	.....

1949 figures are provisional.

Rates equal number of deaths under 1 year per 1,000 live births.

TABLE 2

MATERNAL DEATHS SHOWING NUMBER AND RATES
LOUISIANA, 1925, 1930, 1935, 1940, 1945, 1949

Year	Total	Rate	White	Rate	Nonwhite	Rate
1925	464	11.1	219	8.3	245	15.8
1930	414	9.7	208	8.0	206	12.3
1935	334	7.9	158	6.5	176	9.9
1940	270	5.3	112	3.7	158	7.5
1945	137	2.4	63	1.8	74	3.3
1949	91	1.2	31	0.7	60	2.1

1949 figures are provisional.

Rates equal number of deaths per 1,000 live births.

TABLE 3

NEONATAL DEATHS SHOWING NUMBER AND RATES
LOUISIANA, 1925, 1930, 1935, 1940, 1945, 1949

Year	Total	Rate	White	Rate	Nonwhite	Rate
1925	1469	46.1	768	39.5	701	56.6
1930	1645	38.4	849	32.5	796	44.9
1935	1600	37.9	784	32.1	816	45.7
1940	1869	36.7	861	28.8	1008	47.9
1945	1768	30.4	880	24.8	888	39.3
1949	1989	27.3	.....	.....	.....	.....

1925 figures exclusive of Orleans Parish.

1949 figures are provisional.

Rates equal number of deaths under 1 month per 1,000 live births.

Louisiana State Department of Health, Division of Public Health Statistics Tabulation and Analysis Section. March 21, 1950.

TABLE 4

LEADING CAUSES OF MATERNAL DEATH
LOUISIANA, 1925

Cause	Number	Rate
Puerperal albuminuria and convulsion	172	4.1
Puerperal septicemia	85	2.0
Other accidents of pregnancy	64	1.5
Abortion	54	1.3
Puerperal hemorrhage	40	1.0
Other and unspecified conditions of childbirth and the puerperium	24	0.6
Cesarean section	6	0.1
Other surgical operations ond instrumental delivery	6	0.1
Ectopic gestation	5	0.1
Puerperal phlegmasia alba dolens, embolus, sudden death	5	0.1
Other conditions following childbirth (not otherwise defined)	3	0.1

Rates equal number of deaths per 1,000 live births.

Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950.

TABLE 5

LEADING CAUSES OF MATERNAL DEATH
LOUISIANA, 1930

Cause	Number	Rate
Puerperal albuminuria and convulsion	157	3.7
Puerperal septicemia	109	2.5
Puerperal hemorrhage	49	1.1
Other accidents of pregnancy	39	0.9
Abortion	13	0.3
Puerperal phlegmasia alba dolens, embolus, sudden death	12	0.3
Ectopic gestation	9	0.2
Cesarean section	9	0.2
Other and unspecified conditions of childbirth and the puerperium	8	0.2
Following childbirth (not otherwise defined)	8	0.2
Other surgical operations and instrumental delivery	1	0.0

Rates equal number of maternal deaths per 1,000 live births.

Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 6

LEADING CAUSES OF MATERNAL DEATHS
LOUISIANA, 1935

Cause	Number	Rate
Puerperal septicemia and pyemia	89	2.1
Puerperal albuminuria and eclampsia	71	1.7
Puerperal hemorrhage	37	0.9
Abortion with mention of septic conditions	26	0.6
Accidents of childbirth (excluding Cesarean operation)	21	0.5
Other toxemias of pregnancy	21	0.5
Abortion without mention of septic conditions (to include hemorrhage)	17	0.4
Cesarean operation	16	0.4
Ectopic gestation without mention of septic conditions	13	0.3
Other accidents of pregnancy (not to include hemorrhage)	10	0.2
Puerperal phlegmasia alba dolens, embolus, sudden death (not specified as septic)	9	0.2

Rates equal number of deaths per 1,000 live births.
Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 7

LEADING CAUSES OF MATERNAL DEATHS
LOUISIANA, 1940

Cause	Number	Rate
Infection during childbirth and the puerperium	58	1.1
Toxemias of pregnancy	46	0.9
Hemorrhage of childbirth and the puerperium	37	0.7
Abortion with mention of infection	31	0.6
Abortion without mention of infection	25	0.5
Puerperal toxemias	24	0.5
Other accidents and specified conditions of childbirth	14	0.3
Ectopic gestation	12	0.2
Other and unspecified conditions of childbirth and the puerperium	11	0.2
Other diseases and accidents of pregnancy	8	0.2

Rates equal number of deaths per 1,000 live births.
Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 8

LEADING CAUSES OF MATERNAL DEATHS
LOUISIANA, 1945

Cause	Number	Rate
Other accidents and specified conditions of childbirth	22	0.4
Hemorrhage of childbirth and the puerperium	21	0.4
Puerperal toxemias (excluding death before delivery)	21	0.4
Abortion with mention of infection	18	0.3
Toxemias of pregnancy (death before delivery)	18	0.3
Infection during childbirth and the puerperium	17	0.3
Abortion without mention of infection	9	0.2
Ectopic gestation	5	0.1
Other and unspecified conditions of childbirth and the puerperium	3	0.1
Other diseases and accidents of pregnancy (death before delivery)	2	0.0
Hemorrhage of pregnancy (death before delivery)	1	0.0

Rates equal number of maternal deaths per 1,000 live births.
Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 9

LEADING CAUSES OF MATERNAL DEATHS
LOUISIANA, 1948

Cause	Number	Rate
Puerperal toxemias (excluding death before delivery)	24	0.3
Toxemias of pregnancy (death before delivery)	21	0.3
Hemorrhage of childbirth and the puerperium	17	0.2
Infection during childbirth and the puerperium	17	0.2
Ectopic gestation	9	0.1
Diseases and accidents of pregnancy without mention of infection (death before delivery)	7	0.1
Abortion without mention of infection	6	0.1
Other and unspecified conditions of childbirth and the puerperium	5	0.1
Hemorrhage of pregnancy (death before delivery)	4	0.1
Other specified conditions of childbirth	4	0.1
Abortion with mention of infection	3	0.0
Laceration, rupture, or other trauma of pelvic organs and tissue	2	0.0
Diseases and accidents of pregnancy with mention of infection (death before delivery)	1	0.0

Rates equal number of deaths per 1,000 live births.
Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 10

MATERNAL DEATHS SHOWN BY AGE
LOUISIANA, 1925, 1930, 1935, 1940, 1945, 1949

	1925	1930	1935		1940		1945	1949
Total	464	414	334	Total	270	Total	137	92
10-19	93	74	49	10-14	2	15-19	13	10
20-29	194	186	137	15-34	217	20-29	53	38
30-39	143	126	116	35 & over		30-39	58	37
40-49	33	28	32	.....	.....	40-49	13	6
Unknown	1	.....	.....	Unknown	.....	Unknown	.....	1

All 1949 figures are provisional.
Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 24, 1950

TABLE 11

PERCENTAGE OF LIVE BIRTHS BY ATTENDANT
PERCENTGE OF INFANT AND MATERNAL DEATHS
LOUISIANA, 1925, 1930, 1935, 1940, 1945, 1948

	LIVE BIRTHS					INFANT DEATHS		MATERNAL DEATHS	
Year	Total	Attended by doctor	%	Attended by other	%	Number	%	Number	%
1925	41902	23194	55.4	18708	44.6	3816	9.1	464	1.1
1930	42888	24657	57.5	18231	42.5	3356	7.8	414	1.0
1935	42248	25463	60.3	16785	39.7	2829	6.7	334	0.8
1940	50973	35789	70.2	15184	29.8	3187	6.3	270	0.5
1945	58064	45557	78.5	12507	21.5	2507	4.3	137	0.2
1948	73769	62578	84.8	11191	15.2	2807	3.8	120	0.2

Percents shown for infant and maternal deaths are the percent of total live births.
Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 12

LEADING CAUSES OF INFANT DEATHS
LOUISIANA 1925

Cause	Number	Rate
Diarrhea and enteritis (under 2 years)	829	19.8
Premature birth	640	15.3
Congenital debility	507	12.1
Bronchopneumonia	313	7.5
Congenital malformations	167	4.0
Injury at birth	132	3.2
Influenza	128	3.1
Syphilis	127	3.0
Whooping cough	119	2.8
Lobar pneumonia	101	2.4

Rates equal number of deaths under 1 year per 1,000 live births.
Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 13

LEADING CAUSES OF INFANT DEATHS
LOUISIANA, 1930

Cause	Number	Rate
Premature birth	689	16.1
Diarrhea and enteritis (under 2 years)	419	9.8
Bronchopneumonia	332	7.7
Injury at birth	197	4.6
Influenza	152	3.5
Other diseases peculiar to early infancy	135	3.1
Congenital debility, icterus and sclerema	132	3.1
Syphilis	110	2.6
Lobar pneumonia	85	2.0
Congenital malformations of the heart	8	1.9

Rates equal number of deaths under 1 year per 1,000 live births.
Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 14

LEADING CAUSES OF INFANT DEATHS

LOUISIANA, 1935

Cause	Number	Rate
Premature birth	774	18.3
Bronchopneumonia (including capillary bronchitis)	319	7.6
Diarrhea and enteritis (under 2 years)	288	6.8
Injury at birth	188	4.4
Congenital malformations (stillbirths not included)	123	2.9
Congenital debility	112	2.7
Syphilis	108	2.6
Other diseases peculiar to early infancy	105	2.5
Influenza	102	2.4
Lobar pneumonia	99	2.3

Rates equal number of deaths under 1 year per 1,000 live births.

Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 15

LEADING CAUSES OF INFANT DEATHS

LOUISIANA, 1940

Cause	Number	Rate
Premature birth	900	17.7
Bronchopneumonia	368	7.2
Diarrhea, enteritis, and ulceration of the intestines (under 2 years)	285	5.6
Injury at birth	217	4.3
Other diseases peculiar to the first year of life	179	3.5
Influenza	163	3.2
Congenital malformations	149	2.9
Syphilis	111	2.2
Congenital debility	110	2.2
Whooping cough	96	1.9

Rates equal number of deaths under 1 year per 1,000 live births.

Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 16

LEADING CAUSES OF INFANT DEATHS

LOUISIANA, 1945

Cause	Number	Rate
Premature birth	797	13.7
Bronchpneumonia	257	4.4
Injury at birth	201	3.5
Congenital malformation	197	3.4
Diarrhea, enteritis, and ulceration of the intestines (under 2 years)	162	2.8
Other diseases peculiar to the first year of life	161	2.8
Influenza	85	1.5
Congenital debility (cause not stated)	70	1.2
Lobar pneumonia	66	1.1
Unclassified pneumonia	50	0.9

Rates equal the number of deaths under 1 year per 1,000 live births.

Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 17

LEADING CAUSES OF INFANT DEATHS

LOUISIANA, 1948

Cause	Number	Rate
Premature birth (cause not stated)	970	13.1
Bronchopneumonia	336	4.6
Congenital malformations	265	3.6
Injury at birth	256	3.5
Other diseases peculiar to the first year of life	210	2.8
Diarrhea, enteritis and ulcerations of the intestines	114	1.5
Lobar pneumonia	64	0.9
Influenza	63	0.9
Congenital debility (cause not stated)	54	0.7
Syphilis	38	0.5

Rates equal number of deaths per 1,000 live births.
Louisiana State Department of Health
Division of Public Health Statistics
Tabulation and Analysis Section
March 21, 1950

TABLE 18

STILLBIRTHS BY RACE SHOWING RATES

LOUISIANA 1920-1948

Year	Total	Rate	White	Rate	Nonwhite	Rate
1920**	1,399	37.6	*	*	*	*
1921**	1,516	38.9	714	39.1	802	75.3
1925	2,204	52.6	1,048	39.8	1,156	74.3
1928	2,451	58.4	1,075	41.7	1,376	85.2
1930	2,294	53.5	1,005	38.5	1,289	76.8
1935	2,099	49.7	847	34.6	1,252	70.4
1940	1,938	38.0	793	26.5	1,145	54.3
1945	1,692	29.1	720	20.3	972	43.0
1948	1,883	25.5	762	16.9	1,121	39.2

Rates equal number of stillbirths per 1,000 live births.
**Does not include Orleans figures.
*Not available.

DISCUSSION

Dr. Curtis J. Lund (New Orleans): From time to time, it is good to stop and see what kind of job we are doing. Dr. Martin has done that and has indicated that we have been doing at least an improved job as far as maternal and infant mortality are concerned. However, it is not the purpose of this discussion to present a cause for mutual admiration. On the contrary, it is important that we consider where, in the future, improvement can be made.

I think you can see from the rapid improvement in the rate of maternal mortality and the rather low rate which exists at the present time that it is no longer the valuable criterion of good obstetrics that it was fifteen or twenty years ago. There should be very few maternal deaths at the present time.

On the other hand, we have a much better criterion in evaluation of obstetrics and that is the neonatal mortality, and here we find the improvement is not so great and that the need for further improvement is real. If we could have seen the charts that Dr. Martin presented, we would have seen more clearly the fact that infant mortality has dropped tremendously the past twenty-five years but neonatal mortality has not kept pace. In other words, babies still die too frequently under one month of age.

It is possible to reduce the maternal mortality to a low rate for, as in Charity Hospital at the present time, the maternal mortality rate has dropped from 2 per thousand live births in 1945 to .05 per thousand in 1949. Such a mortality rate was obtained from services delivering at least 75 per cent of nonwhite mothers. This shows definitely that the rate for the nonwhite can be reduced to a rate comparable to that of the white.

There are, I think, three spheres in which we can do much in the prevention of maternal and neonatal mortality. I have taken statistical liberties with Dr. Martin's paper and have attempted to lump some of the causes of death together. For example, it is certainly obvious that a good many of the deaths are associated with hemorrhage. We find that one-third of maternal deaths are due to hemorrhage. Another third are due to toxemia. The remaining third then are due to infections and a host of other associated diseases.

Heart disease at the present time should probably be the leading cause of death in a well regulated practice. We still have deaths from hemorrhage, infection, and toxemia exceeding those from heart disease.

What can we do about reducing deaths from toxemia? I do not believe there is anything to be said except that it reflects almost always the state of prenatal care. Adequate prenatal care will materially reduce the death rate from toxemia. This does not mean prenatal care in name only. Secondly, there must be active treatment of toxemia and not inactive or procrastinating treatment of toxemia. Certainly active treatment means something more than instruction to the patient that she should not eat so much salt.

Too many women are dying of blood loss. I hope that the time will come, and I think it is coming fairly soon, when a physician will be guilty of malpractice if he has lost a patient from bleeding because blood was not available. Blood banks must be established and must be available to anyone and everyone who is doing obstetrics. There is no place in medicine where it is needed so vitally as in obstetrics. Much can be done in prenatal care. Recognition and treatment of anemia will go far in reducing the hazards of hemorrhage. Routine Rh and ABO typing should be done in advance so that blood can be available at a moment's notice when the emergency occurs.

A third sphere of our efforts should be directed towards prematurity. The wastage of life associated with prematurity in this state is appalling. In 1949, 970 babies died with conditions surrounding premature birth. The number exceeds deaths from tuberculosis, or chronic rheumatic heart disease, or automobile accidents. An active campaign to reduce the incidence of premature birth and an active campaign to save more premature babies is clearly indicated. The very fact that Dr. Martin stated prematurity death rate has dropped from 15 to 13 is clear evidence that little has been done. Thirteen deaths per thousand prematures is very high.

What can be done? It has been shown that the incidence of premature births which ranges from 8 to 10 per cent in most areas can be reduced to 3 or 4 per cent in well regulated practice. Prenatal care will reduce the incidence of premature delivery. Secondly, we have need for better care of the labor of women who are delivering premature infants and, finally, the need for better care of premature babies once they have been born.

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SUDDEN AND UNEXPECTED DEATH IN INFANTS*

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NEW ORLEANS

There is probably no experience more distressing to family and physician alike than a sudden and unexpected death in an apparently healthy infant. Invariably, parents are haunted by feelings of possible

*Presented at the Seventieth Annual Meeting of the Louisiana State Medical Society, April 26, 1950, in Baton Rouge.

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guilt and search for responsible errors of omission or commission. Understandably, the doctor is plied with questions and pressed for a definite diagnosis. In a vast majority of instances no plausible explanation is apparent and as a consequence it is little wonder at such a time that terms such as status thymico lymphaticus, lymphatism, accidental mechanical suffocation, asphyxia, or anaphylaxis are seized upon. However convenient and apparently suitable such labels may seem to be in the certification and explanation of such deaths, their use and misuse has led to deeply rooted misconceptions and misinterpretations. It has been only within the last few years that some clarification of the problem has been possible through accumulated autopsy material. Specifically, reference is made to careful examination of representative tissues by skilled pathologists who have not only been interested in the problem, but have been unwilling to take anything for granted. When such a study is made, significant causes of death are found in up to 85 per cent of these "unexplained" cases.

It should be emphasized that a sudden death during the course of a known disease is neither unusual nor difficult to explain. As has been pointed out by Farber[1], and Gellis[2], the real problem lies in completely unexpected sudden deaths which occur in an appreciable number of infants and small children. The following discussion is concerned primarily with such unexpected sudden deaths.

COMMON MISCONCEPTIONS

Implication of the thymus gland in sudden and unexpected death is not new. In 1615, Plater described tracheal compression as a cause of death, and referred to the condition as *Mors Thymica*. From such beginnings an almost continuous controversy has been maintained in the medical literature up to the present time. Not many years ago prophylactic or therapeutic irradiation of the thymus during the newborn period was undertaken with great frequency. In fact, such treatment was widely recommended as a safeguard against anesthetic or other unexpected death in association with tonsillectomy and adenoidectomy. Although it is certainly true that the size of the gland can be markedly reduced by this practice, and probably true that the amount of irradiation commonly used did not adversely affect the patient, it does not follow that such procedures are justifiable. In the light of our present knowledge, thymic irradiation has been almost completely abandoned.

In 1945, an exhaustive treatise by Carr[3] on status thymico lymphaticus appeared in the pediatric literature. This author supports the view that, in some instances, abnormalities of the thymus are related to unexpected death, but states that the mechanism is of secondary importance from a numerical viewpoint. On the other hand, most professional opinion including that of Goldbloom,[4] Farber,[5] and Werne and Garrow[6] indicates that the lymphatic constitution or status thymico lymphaticus is not a pathologic entity, as Carr believes, but rather that other significant causes of death can be found if detailed and appropriate studies are completed.

Much of the confusion concerning the pathologic potential of the thymus stems from our startling ignorance of its normal function. Various workers have suggested relations between this glandular structure and each of the following: somatic growth, parathyroid function and calcium metabolism, adrenal function both cortical and medullary, gonadal development and fecundity, osseous development and epiphyseal closure, general nutrition, and even intelligence! Thymic enlargement has been reported repeatedly in exophthalmic goiter, Addison's disease, acromegaly, adrenal hypoplasia, anencephaly, rickets, and myasthenia gravis. Many investigators have attempted to clarify this confusing situation, but have been unable to relate their observations to definite single or multiple functions of the gland. Thymic extracts have been studied in experimental animals with indifferent and inconclusive results. Exhaustive studies of thymic size, position, and weight have been made in all age

groups and in various states of health and disease. From the latter, some general conclusions can be drawn. In the first place, the gland obviously varies within wider ranges of size and weight than does any other organ of the body. Without question it is largest during the periods of most rapid growth during childhood. Following puberty there is progressive involution of the gland with replacement by fat and fibrous tissue. Finally, in old age its weight again approximates that in the newborn. In states of malnutrition the weight is commonly one third less than that found if optimal nutrition obtains, and both size and weight decrease very rapidly during acute illness. It has even been suggested that the thymic weight of individuals living in warm climates is less than that found in persons from colder areas. In a newborn infant the generally accepted thymic weight is 10 to 12 grams. This is ordinarily doubled at one year and trebled at twelve years.[7]

In relation to the problem of sudden unexpected death in infancy the position of the gland in the anterior mediastinum is of at least equal importance with its size and weight. Caffey[8] has pointed out that the broadened mediastinal shadow of young infants is most logically indicative of a thin superficially placed gland which has not been compressed and moulded posteriorly between fully expanded lungs. He has further shown that the supracardiac shadow becomes widened during expiration. It follows, then, that the mere finding of a broad thymic shadow in the usual roentgenogram of the chest cannot be interpreted as demonstrating enlargement of the gland or the probability of its encroachment upon the trachea. Carr points out that the thymus may extend downward sufficiently to overlap the auricles of the heart, or in similar fashion exert pressure upon the recurrent laryngeal nerve, thereby interfering with cardiovascular function, or possibly mediating abnormal vagal effects. Since the heart operates normally in relation to contiguous structures it is difficult to conceive of a thin lapette of thymic tissue seriously and suddenly interfering with cardiac function. The second hypothesis is entirely speculative, and, like the first could only be applied in those few instances in which no other significant cause of death was demonstrable and in which the specific anatomical relationship in question could be unmistakeably identified. Farber studied the thymus in 2000 consecutive autopsies and concluded that, in no case, could the gland be considered pathological in size, although large glands were occasionally encountered in children who had experienced no symptoms referable to thymic enlargement. Significant causes of death, exclusive of the thymus were found in all cases. Finally, considering the soft pliable character of the thymus as compared to the relative rigidity of the trachea, compression of the latter by the former would seem highly improbable.

A syndrome, status thymico lymphaticus, is described as including several characteristic stigmata. The first is so-called thymic hyperplasia, the pathological reality of which can be logically questioned. Secondly, generalized lymphoid hyperplasia is emphasized, a finding which would be difficult to assess in the childhood age group in which hypertrophy of lymphoid tissues is so frequently encountered. Thirdly, examples of the syndrome are said to evidence a flabby overgrowth of fat. Realizing the increased size of the gland in heavy, well nourished children this feature is not impressive. Fourthly, there is bone marrow hyperplasia. This too has little meaning unless in reference to specific patients because of the relative frequency of the anemias of childhood and the hyperplastic reaction of the marrow in attempted correction. Finally, the vague term, hypoplasia of the heart and great vessels, is included, but in the absence of adequate definition cannot be intelligently discussed. In brief, pathology of the thymus gland leading to sudden and unexpected death must be viewed as a most improbable and infrequent possibility and one which most workers in the field are unwilling to accept.

In recent years much attention has been

focussed upon accidental mechanical suffocation in infancy. This reawakened interest, concomitant with the onset of World War II, is logically relatable to concern for the infant population incident to the disruption of families and their permanent residences; together with the relatively high birth rate, and the frequency of maternal employment outside the home. Further one can speculate that during the war years a paucity of civilian medical personnel and the extreme case loads carried by those available did not permit the careful and critical evaluation of sudden unexpected deaths. In any event certification of death as due to accidental mechanical suffocation became alarmingly frequent and led the Metropolitan Life Insurance Company in 1942 to describe it as the greatest hazard of infancy. Abramson,[10] in 1944, accepting the diagnosis as listed on death certificates and working from obtainable historical data found the peak incidence in late fall and early winter, and the highest percentage (85 per cent) occurring in infants under six months of age. Sixty-eight per cent were male and 31 per cent female. The types of smothering agents such as bedclothes, pillows, mattresses, mother's breast or arm were tabulated, and because 68 per cent of his cases were found in the face-down position, this attitude was categorically denounced as dangerous. Reviewing the subject in 1945 Wooley[9] observed that suffocation as a listed cause of death was more frequent than the mortality of measles, diphtheria, and scarlet fever combined, during a ten year period. The danger of mechanical suffocation stimulated editorials in leading medical journals and soon led to discussions in lay publications.

Not the fact, but the disproportionate frequency of suffocation led such investigators as Davison, Werne and Garrow, Wooley and others to study the problem. The coincidental peak incidence of both accidental mechanical suffocation and upper respiratory disease in late fall and winter months was pointed out. The paradox of finding that the greatest number of suffocated infants was in the three to four month age group, and not in the neonatal period when weakness and helplessness is most marked, became apparent. Because sudden death in infancy was made legal cause for autopsy in England, Davison was able to report 318 cases studied between 1938 and 1944. Thirty-eight were due to mechanical suffocation. Overlying while in the bed of another person was established in 18 instances; the aspiration of vomitus in 11; suffocation while sleeping alone in 6; and unknown factors in 3. Similar studies in this country by Werne and Garrow showed an even more striking reduction of the number of established cases of suffocation, in spite of the fact that the diagnosis had been made much more frequently than in the preceding decade (57 per cent increase).

Attempts to maintain asphyxial obstruction of the nose and mouth are most difficult since immediate resistive movements are made by all but the weakest and most debilitated infants. Realizing this fact and further recalling the difficulty in maintaining high oxygen concentrations within presumably impermeable tents, Wooley attempted to determine oxygen and carbon dioxide concentrations under conditions of simulated suffocation with bedclothes, pillows and mattresses. Without resorting to the expedient of carefully fastened rubber sheeting over the infant's head he was unable to appreciably affect the relative concentrations of gases at the subject's nose and mouth. Accumulated evidence, therefore, justifies the conclusion that mechanical suffocation from extrinsic agents is exceedingly unusual, although rare instances of garrottement by patented sleeping garments, strangulation between poorly spaced or defective crib bars, and particularly overlying by another person do occur. As a cause of sudden death aspiration of foreign material whether amniotic fluid, formula, vomitus, excessive secretions, or foreign bodies is not uncommon. In this connection consideration of the face-down position is pertinent. Adams[11] has pointed out important advantages of this posture from

a physiological viewpoint, in that it allows dependent drainage of secretion from the tracheo-bronchial tree into the oropharynx. Its exaggerated importance in the causation of mechanical suffocation has already been pointed out. Finally the alternative supine position is associated with increased danger of aspirating secretions or other fluids pooled in the posterior pharynx. At least it can be said that one position is no more dangerous than the other with regard to mechanical suffocation.

In connection with accidental mechanical suffocation, the tragic remorse and self incrimination of parents is in itself sufficient stimulus for painstaking and critical autopsy examination. Certainly, the physician is justified in rejecting this diagnostic possibility until and unless microscopic studies eliminate other causes of death. Circumstantial evidence, however suggestive, is not enough.

IMPORTANT MECHANISMS OF DEATH

Reference has been made previously to significant pathology revealed at autopsy in deaths incorrectly attributed to either status thymico lymphaticus or accidental mechanical suffocation. By far the most common is infection. For purposes of discussion this can be divided into two categories; first pneumonitis and second generalized sepsis. With respect to the former Werne[12], referring to a ten year study consisting of 50 cases of unexpected death in infancy states, "The majority of sections of lung showed the occurrence of bronchitis, peribronchitis, and peribronchial pneumonitis, mononuclear cells predominating", and further that "extreme capillary congestion" and "pericapillary and intra-alveolar edema" were characteristic. Adams encountered essentially similar findings in cases of virus pneumonitis among newborn. Labate[13] in a study of fetal and neonatal mortality at the Bellevue Hospital in New York, states that 39.3 per cent of neonatal deaths were due to pulmonary lesions. Under this classification he lists congenital (intrauterine) pneumonitis, aspiration pneumonia, and bacterial pneumonia, as well as pulmonary hemorrhage, and hyaline membrane. Miller[14] believes that the hyaline membrane referred to above is phagocytized by polymorphonuclear and mononuclear cells and that this combined appearance constitutes the chief form of pneumonia in the newborn infant. The hyaline membrane itself he attributes to pulmonary inflammation antedating delivery. Pulmonary inflammation in infancy may lead to unexpected death, in that definite physical signs are notoriously absent and that infection may pursue an extremely rapid course. Also, the coverings of small infants may conceal evidences of respiratory distress from untrained and uncritical observers.

Pneumothorax occurs with appreciable frequency in the pediatric age group and may or may not be associated with pulmonary disease. Emphysematous blebs are commonly associated with diffuse bronchopneumonia, and if these are superficial, rupture into the pleural cavity may occur. The added insult of pneumothorax as a complication of pulmonary infection can be sufficient to cause death. In some instances a valvelike mechanism obtains allowing air to enter but not leave the pleural space. In consequence, a tension pneumothorax with far more serious implications than in the case of simple pneumothorax results. Entirely comparable circumstances may exist in the absence of infection, due to the fact that emphysematous blebs may occur as a development aberration. The effects of their rupture, or so-called spontaneous pneumothorax, occurring, for example, at times of severe crying or choking are indistinguishable from those previously mentioned. Trauma to the bony thorax, such as a fractured rib puncturing the lung, is a rare cause of air accumulation in the pleural cavity during infancy. Finally pneumothorax may complicate tracheotomy, since the delicate deep cervical fascia may become torn at any time after surgery and the insertion of a tracheal canula. Fortunately this is not a common occurrence and it can usually be identified before serious danger has developed.

In 1934 Farber stressed the importance of fulminating infections in sudden death of infancy. In his cases the streptococcus was the most commonly incriminated organism, although staphylococci, pneumococci, meningococci, and dysentery bacilli could also be incriminated. We now realize that the bacterial spectrum is considerably wider but the concept of rapidly fatal sepsis has been repeatedly confirmed. Progress of initial bacteremia may be so swift that localization in any one organ is not impressive. However early interstitial bronchopneumonia with widespread petechial hemorrhage throughout the lungs is an almost invariable accompaniment. Cerebral edema, edema of the optic nerve, and of the spinal cord, as well as petechiae of the heart, kidneys and liver, probably the result of venous thrombi, are prominent. An antecedent history of recognizable illness is conspicuously absent, and, as Farber observes, most of these patients are signed out as thymic deaths or mechanical suffocations after efforts at resuscitation have been to no avail.

In addition to bacterial infections, those of virus etiology may exhibit fulminant and rapidly fatal courses. For example, death has been known to occur in less than twenty-four hours in eastern equine encephalitis, and in fact occurs in four days or less in approximately 60 per cent of all persons acquiring the disease.

Adrenal cortical damage commonly found in association with generalized sepsis is worthy of special mention. Bilateral adrenal hemorrhage in meningococcal infections has long been recognized as the Waterhouse-Fridrichsen syndrome, and in the opinion of some a similar appearance is seen in fulminant infections caused by other bacterial agents. In addition Rich[15] described the frequent occurrence of tubule-like formations from the splitting of cell cords in the zona fasciculata and glomerulosa of the adrenal cortex in streptococcal, pneumococcal and diphtheritic infections. These formations have a resemblance to renal tubules containing serum, fibrin, polymorphonuclear leucocytes, and necrotic cortical cells. The author realizes that cardiac failure and the effects of bacterial products independent of adrenal failure are valid explanations of circulatory collapse in some instances, but is convinced that clinical evidences of sudden collapse and death are closely correlated with the pathology described in many patients.

The prominence of congenital anomalies of the heart and great vessls in unexpected sudden death is rather difficult to determine. We know that such lesions are found in approximately 4 per cent of autopsies in hospitals caring for infants and children, as compared to an incidence of approximately 1 per cent in general hospitals. However, we also know that many of these lesions are compatible with life beyond infancy. Further, it is usually possible to suspect the presence of congenital heart disease even though the anatomical diagnosis is obscure, in which case sudden death can hardly be considered as unexpected. Nevertheless, it is conceivable that unexpected death in the neonatal period could result from pulmonary artery atresia, complete transposition of the great vessels, persistence of primitive aortic arches as a vascular ring, anomalies of the coronary circulation, and finally coarctation of the aorta. It is significant that Farber considers congenital anomalies of the cardiovascular system as one of the principal causes of sudden death in early life.

The broad subject of hemorrhage can be conveniently divided into intracranial hemorrhage, pulmonary bleeding, and bleeding into specific organs or body cavities. The problem of bleeding within the cranial vault consequent to compression and moulding of the fetal head during delivery is widely understood. The most extensive hemorrhages follow tears in the tentorium and falx, or severance of the vein of Galen at the point of its attachment to the tentorium. Rupture of small cortical veins is also frequent as a cause of subarachnoid bleeding. Although the obstetrical history and clinical appearance of the patient often suggests the diagnosis prior to death, this is not necessarily

true. Not infrequently an infant who is apparently normal at the time of delivery will be found in critical condition a few hours later. Entirely comparable circumstances may obtain following trauma or violence at any time after birth. This possibility must be borne in mind even in the absence of external signs of head injury or clear-cut history of trauma. Of course, in the presence of hypoprothrombinemia, often referred to as hemorrhagic disease of the newborn, bleeding may bear no relation to the application of physical forces and similarly be independent of other pathology.

Two relatively infrequent causes of intracranial hemorrhage remain. The first of these is bleeding into a vascular brain tumor. The largest number of central nervous system neoplasms are subtentorial, and though ultimately producing increased intracranial pressure together with more or less localizing neurologic signs, may not do so until late in the clinical course. Because of the position of many of them in proximity to vital centers of the brain stem extensive hemorrhage into their substance can be rapidly fatal. Finally, congenital aneurysms at points of branching in the circle of Willis must be considered. Such vascular abnormalities are variously estimated to occur in 5 to 10 per cent of all individuals. They rarely give rise to difficulty in the period of infancy unless intracranial arterial hypertension is also present. It was in this connection that coarctation of the aorta was mentioned previously as a contributory cause of unexpected death. This lesion located distal to the origin of the cranial arterial blood supply produces sufficient increase in blood pressure to endanger weakened aneurysmal dilatations in the cerebral circulation.

Pulmonary hemorrhage is a less common explanation of sudden death than is intracranial hemorrhage, but is listed sixth or seventh in point of frequency by some investigators.[16] Anoxia during delivery is known to incite exaggerated respiratory efforts in the fetus. These in turn effect relatively high negative intrathoracic pressures under conditions of complete airway obstruction. As a consequence small thin walled pulmonary vessels may rupture, and resultant bleeding reduce available alveolar surfaces to a critical degree within a matter of one or more hours. It should be borne in mind that the presence of pulmonary hemorrhage associated with other types of pathology in the lungs may be an important contributory though not the sole mechanism of death. The frequency of this type of hemorrhage in association with fulminant sepsis has already been pointed out.

A third category of hemorrhage is that occurring within the abdominal cavity. As an unusual and extremely occult complication of manipulating the infant during delivery, or of overzealous and ill-chosen resuscitative attempts, subcapsular hemorrhage of the liver is encountered. Subsequent handling in the course of daily care and feeding occasionally results in rupture of the encapsulated collections before organization has occurred. Release of pressure within the hematoma allows recurrence of free bleeding from the primary site, usually a laceration in the liver itself, and rapid exsanguination into the peritoneal cavity results. As would be suspected this diagnosis is usually made at autopsy.

Death of the type which concerns us here has long been known to occur in intimate association with surgical procedures. Because tonsillectomy and adenoidectomy are the most frequent operation undertaken in the United States today, unexplained fatalities have been particularly associated with these technics. Careful surveys, however, do not bear this out. In fact a recent report from the Children's Hospital of Washington, D. C.[17] shows a greater occurrence rate with abdominal and neurological procedures than with operations on the oropharynx and ears. No single mechanism can be incriminated in all instances, rather any one of several acting alone or in combination offer the explanation. Important among these is choice of unsuitable anesthesia and its improper administration. Local as well as general anesthetic agents must be considered because of the possi-

bility of anaphylaxis consequent to their use. Similarly sensitivities to atropine as well as to other drugs whether given preoperatively or not are observed with considerable frequency in early life and explain some fatalities. Particularly distressing are those deaths following, after twenty-four to forty-eight hours, the uneventful completion of elective and often minor surgery. Careful autopsy examination in the majority of these cases reveals overwhelming sepsis in no way differing from that mentioned previously but presumably having the operative site as the portal of entry. The aspiration of foreign material or blood by an anesthetized patient is no longer as extreme a danger as it was in the past because the possibility of its occurrence is generally recognized and technics, such as proper postural drainage and bronchoscopy, are better understood and more widely applied. Finally abnormal sensitivity of vago-vagal reflexes including those mediated by the carotid sinus must be mentioned. Abnormal positions and tensions on the neck, as well as repeated palpation of the carotid pulse of an anesthetized patient are possibilities in the excitation of these reflex effects. Suffice it to say that proof of their occurrence is most difficult to obtain, although circumstances in selected instances are highly suggestive.

SUMMARY AND CONCLUSIONS

No attempt has been made to consider all possible causes of unexpected sudden death in infancy—just the more important and significant mechanisms, as well as the most common misconceptions have been discussed. Poisonings as with fluoride or cyanide have been omitted because of their exceeding rareness. Anaphylactoid deaths occurring in eczematous infants are not sufficiently numerous or well described to merit attention. Finally the predictable possibility of death, though relatively sudden in character, as seen in acute laryngotracheal bronchitis, justified its exclusion from a discussion of this sort.

A consideration of these tragic happenings in infancy and early chilhood clearly emphasizes the crucial importance of complete and detailed study in each case.

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FRIEDLÄNDER AEROGENES INFECTIONS IN INFANCY*

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In 1882, Friedländer[1] isolated a gram negative encapsulated bacillus from the lungs of 8 patients dying of pneumonia. Since then, the organism which bears his

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Presented at the Seventieth Annual Meeting of the Louisiana State Medical Society, April 26, 1950, in Baton Rouge.

name has been regarded largely as a respiratory pathogen despite many published cases of nonpulmonary disease. This concept prevailed until 1937 when Baehr, Swartzman, and Greenspan[2] reported 198 cases of Friedländer infections. In only 25 instances was the organism implicated in upper or lower respiratory disease, and biliary, peritoneal, genito-urinary infections constituted the majority of their cases. These authors regarded the Friedländer bacillus, when present in the respiratory tract, primarily as a secondary invader of previously diseased pulmonary tissue.

It is now abundantly clear that infections caused by these organisms may involve any part of the body and may produce a wide variety of pathological lesions. Nevertheless, the reported incidence of Friedländer infections remains low. It would appear that a more careful bacteriological study might define a higher incidence. The tendency to regard all gram negative bacilli as coliforms and the failure to perform capsular stains probably results in the classification of many Friedländer organisms as members of the coliform group. This error is often perpetuated because of the similarity of some of the fermentation reactions of both groups.

The appearance of several reports, recently, dealing with Friedländer-aerogenes diseases may be indicative of an actual increase in these infections. One group[3] has commented on the striking increase in number and severity of urinary tract infections caused by *Aerobacter aerogenes*. The number of such reports, however, is still too small to justify the conclusion that Friedländer-aerogenes infections are on the increase.

Part of the seeming increase has resulted from the widespread use of penicillin. The administration of this antibiotic eliminates the normal gram positive pharyngeal flora and permits the appearance in predominance of gram negative coliforms.[4] This effect of the antibiotic is responsible for the biologic phenomenon of superinfection which occurs in the course of penicillin therapy. Several instances have now been recorded in which pneumococci, in patients with pneumonia, have disappeared from the pharynx during penicillin therapy without a concomitant clinical cure. Further study revealed the presence of *Aerobacter aerogenes* which accounted for the progression of the disease.[5] Recovery ensued only after the use of streptomycin. The use of penicillin may disturb the relationship by means of which certain of the bacteria are normally held in check.[6] This same phenomenon had not been described with sulfadiazine.

The introduction of many antibiotics, each with a well-defined spectrum of biologic activity, has given bacteriologic identification of organisms practical therapeutic implications. Because some of the newer drugs have been demonstrated to be curative in Friedländer-aerogenes infections, it is now necessary to look for these organisms especially in those patients not responding to the administration of sulfadiazine and penicillin.

Over a period of one year we have encountered 10 patients whose infections were caused by organisms of the Friedländer-aerogenes group. Included among them are 3 with pneumonia, 1 with meningitis, 1 with a urinary tract disease, and 5 with septicemia.

The Friedländer bacillus and *Aerobacter aerogenes* are both gram negative, nonmotile, encapsulated rods and characteristically produce stringy, mucoid colonies. Not only are they morphologically and culturally indistinguishable but they also have very close biochemical and serological similarities. The custom of designating those organisms isolated from the respiratory tract as Friedländer bacilli and those isolated elsewhere as *Aerobacter aerogenes* would apear to have no sound bacteriologic basis.

It has been amply demonstrated that no significant differences between the two organisms are revealed by the use of differential media, by fermentation reactions, or by agglutination and complement fixation methods.[7] The capsule contains a soluble

specific substance closely related to that found in the capsule of type II pneumococcus.[8] Julianelle[9] was able to distinguish three types of Friedländer bacilli designated as A, B, and C, although a great many varieties could not be so classified and were placed in a heterogeneous group X. The same author subsequently showed that there is no correlation between the serologic types and the fermentation reactions and that the fermentation reactions were not reliable in differentiating the Friedländer bacillus from closely allied organisms.

Recent work[10] has emphasized the close immunologic and biochemical similarities between the Friedländer bacillus and *Aerobacter aerogenes* on the one hand, and between these two organisms and *Escherichia coli* on the other. All three organisms may exist in the mucoid, smooth, or rough phase designated as M, S, and R. The Friedländer bacillus and *Aerobacter aerogenes* ordinarily exist in nature in the encapsulated or mucoid phase while *Escherichia coli* is, as a rule, nonencapsulated or in the smooth phase. Under certain conditions, *Escherichia coli* may be encapsulated and the Friedländer bacillus may be decapsulated and produce a smooth colony resembling the coliforms. No clear-cut differences exist when all three are in the same phase.

The organisms of the Friedländer-aerogenes group are ubiquitous and distributed widely in nature. In man they occur in two main sites of localization, in the intestinal and respiratory tracts, where they may exist in the carrier state.

FRIEDLÄNDER PNEUMONIA

Three of our patients had Friedländer pneumonia.

Case No. 1. This patient was a 15 month old male infant who was treated for a left lower lobe pneumonia for two weeks prior to admission and had made no significant response. Shortly after admission he developed generalized convulsions and coma. Because of the desperate clinical picture of an overwhelmingly severe infection, streptomycin and aureomycin were administered empirically. An organism of the Friedländer-aerogenes group was subsequently shown to be present, in predominance, on two successive pharyngeal cultures. Several blood cultures were sterile. He made a slow but progressive recovery with only residual pleural thickening.

Case No. 2. This patient was a 14 month old infant who was admitted to the hospital, after an illness of four days, with bilateral bronchopneumonia. He was acutely ill on admission and in respiratory distress. Clinical response to s ulfadiazine was prompt and dramatic, temperature falling to normal within twenty-four hours. After recovery and discontinuance of sulfadiazine, blood and pharyngeal cultures taken on admission were reported as showing an organism of the Friedländer-aerogenes group.

Case No. 3. This patient, a 6 months old female infant, was ill for three days and on entrance to the hospital had clinical and roentgenographic evidence of bilateral bronchopneumonia. She failed to respond to a single large dose of penicillin, or to penicillin administered every twelve hours for three days. Following identification of a Friedländer-aerogenes organism on pharyngeal culture, streptomycin was administered and the patient made a prompt recovery.

Friedländer's pneumonia, as described for the adult, occurs in both acute and chronic forms and its incidence is considerably higher than in the younger age group. The disease begins as an undifferentiated bronchopneumonia which becomes confluent. There is a pseudolobar distribution with areas of normal lung tissue scattered throughout. A characteristic feature is the widespread tissue necrosis leading to the formation of cavities and abscesses. The abscesses as they appear on the roentgenogram are described as being thin walled. Bacteremia occurs in a large percentage, is transient, and unrelated to mortality. The composite mortality before the advent of streptomycin, as stated by Julianelle,[11] was 82 per cent.

Less than 30 cases of acute Friedländer pneumonia in children have been reported to date. The incidence appears to be low. In children the disease is peculiarly one of infancy, the oldest patient reported having been 15 months of age. Pathologically there is not the same tendency toward necrosis and abscess formation as is seen in adults.

Even in the era before specific chemotherapy, the mortality in children appeared to be lower than in adults. Eleven of the 16 patients, reported before the introduction of the sulfonamides, recovered. Eleven

patients reported subsequently, including the 3 recorded here, all survived. In 9 of the latter group sulfadiazine or penicillin or both proved completely ineffectual, recovery resulting only after the use of streptomycin. In 2 instances of recovery, the only specific therapeutic agent employed was sulfadiazine.

Clinically, the disease is indistinguishable from other bacterial pneumonias and is established only by the isolation of the causative organism. This often comes as a surprise in a patient who is not making the expected progress on sulfadiazine or penicillin. The mucoid tenacious sputum seen so characteristically in adults is not so readily obtained from children. There is nothing typical of the course of the disease or laboratory findings. All grades of severity are encountered.

Some authors[2, 12] are of the opinion that the Friedländer bacillus in its role as a respiratory pathogen is a secondary invader of a previously diseased focus. One need not be concerned with the primacy of these infections. Their importance now lies in the availability of effective chemotherapeutic and antibiotic agents.

MENINGITIS

Meningitis constitutes an important manifestation of extra pulmonary Friedländer-aerogenes disease although it is one of the less frequently reported types.[13] In one series of 705 cases in children observed over a twelve year period only 1 instance of Friedländer meningitis was encountered.[14] In contrast to adults, most of whom have antecedent infection of the upper respiratory tract, children as a rule have no known primary focus.[15] Many of the adults have been diabetics and debilitating disease appears to predispose to meningitis. Positive blood cultures are more serious than in patients with pneumonia as they appear to be associated with a higher mortality. The total number of infants reported to have recovered is 3.

Included among our patients was 1 with meningitis.

Case No. 4. A 5 weeks old male infant entered the hospital because of convulsions, weakness, failure to gain weight, and hydrocephalus. The illness dated from the first week of life. Ventricular puncture revealed purulent fluid from which type C *Klebsiella pneumoniae* was grown on several occasions. Streptomycin was administered intramuscularly over a period of six weeks with gradual restoration of the ventricular fluid to normal. During a second admission for pneumonia, a pneumoencephalogram showed enlarged lateral ventricles with failure to visualize the third and fourth ventricles, thereby confirming the presence of a block which had been suspected clinically. The hydrocephalus was progressive and the patient died nine months later.

GENITO-URINARY DISEASE

The Friedländer-aerogenes group of organisms has always been a common cause of genito-urinary disease. Recently Wilhelm and his coworkers[3] reported an increase in incidence of infections of the urinary tract due to *Aerobacter aerogenes* and indicated that the organism has become more virulent. A survey of the relevant publications of the past twenty years indicates that this group is secondary only to *Escherichia coli* as a common urinary tract pathogen, although Campbell[16] has found the organism only once as a cause of pyelonephritis among 981 cases in children. Some authors regard the urinary tract as the most frequent site of disease following blood stream invasion, localization being facilitated by anatomical malformation. All gradations of severity of the pathological process may be encountered and the organism may establish itself in any part of the urinary tract.[17]

One of our 10 patients belongs in the category of genito-urinary infection.

Case No. 5. This patient, in addition to evidences of Mongolism and congenital heart disease, was jaundiced at birth. Blood culture was sterile and hematologic values were normal. There was no Rh incompatibility. The icterus was thought to be physiologic and the patient was discharged on the fourth day. He was readmitted at 15 days of age because of diarrhea with jaundice still present. The patient presented a continual problem in the maintenance of hydration and acid base equilibrium. His condition deteriorated despite therapy. His kidneys became palpably enlarged, pyuria appeared, and blood and urine cultures were positive for an organism of the Friedländer-aerogenes group. He showed some improvement on sodium sulfadiazine and blood culture became sterile but he expired after a recurrence of dehydration. At autopsy the right kidney was somewhat

enlarged and there was bilateral hydronephrosis and hydroureter with bilateral pyelonephritis.

SEPTICEMIA

We are including 5 patients with septicemia in a separate category although localization appears to have occurred in some. From the point of view of the patient, the septicemia presented the major consideration and localization appeared only as an incident in the general picture.

Mention has already been made of the high incidence of positive blood cultures obtained in patients with Friedländer's pneumonia, of the transient nature of the bacteremia, and of the absence of any positive correlation between the blood stream invasion and mortality. However, in a disease which at one time carried so high a fatality rate it may well be difficult to correlate mortality with any single criterion. Bacteremia is also common in patients with meningitis, in whom it has a serious prognostic significance, but is less frequent in patients with genito-urinary disease.

In cases of Friedländer's pneumonia, the organism invades the blood stream either simultaneously with or secondary to a pulmonary localization. In both meningitis and genito-urinary infection the initial phase is probably a bacteremia with secondary implantation in the meninges and urinary tract. It is difficult as a rule to establish the original source of the organism. Since many of the adults with Friedländer's meningitis have had associated otitic infections, it is quite likely that in this disease the organism comes from an upper respiratory focus. In most cases of urinary tract infection, the source is seldom apparent. Although it has been generally assumed that the organism comes from the bowel, most reports make no mention of recovering the pathogen on stool culture. Once the meningitis and the urinary infections are established, they may become foci which continue to seed the blood stream. It has been indicated that the septicemia may be so overwhelming as to cause death in twenty-four to forty-eight hours without any localization whatever.[18] There have been only a few reports of Friedländer-aerogenes septicemia in infants and children.

We shall now describe briefly the 5 patients with septicemia.

Case No. 6. The patient was a 1470 gram premature infant who developed a severe diarrhea with dehydration and acidosis at six weeks of age. He recovered on standard therapy. Progress was good until three months of age when he became listless and jaundiced. Large numbers of red and white blood cells appeared in the urine. Blood and urine cultures showed a Friedländer-aerogenes organism. Paralytic ileus developed. Therapy consisted of continuous gastric suction and the administration of sulfadiazine, streptomycin, chloramphenicol, blood, and parenteral fluids. Recovery was slow but progressive.

Case No. 7. A three weeks old infant was admitted because of vomiting and diarrhea. Blood culture on admission was sterile. The patient made a good response to a regimen of starvation, parenteral fluids, and transfusion, only to develop a relapse. Jaundice appeared and the patient went progressively downhill and expired. Shortly before death, a throat culture revealed some Friedländer-aerogenes organisms in addition to hemolytic streptococcus and *Staphylococcus albus.* Post mortem cultures of blood and spinal fluid revealed a heavy growth of the Friedländer-aerogenes organism.

Case No. 8. This patient was admitted to the hospital at the age of two days. Birth weight was said to be 1700 grams. His condition was poor from the beginning and he expired after an episode of apnea seven hours after admission. An autopsy was not done. Material taken for bacteriologic study immediately upon death gave the following results: nose and throat cultures revealed *Pseudomonas aeruginosa;* the blood culture was negative, and the spinal fluid contained a pure culture of an organism of the Friedländer-aerogenes group.

Case No. 9. This patient was a premature female infant whose weight on the second day of life was 780 grams. Her condition was critical from the onset. There were episodes of regurgitation and cyanosis and she expired on the fourth day. Cisternal fluid taken immediately on death was grossly bloody. Cultures of the blood and cerebrospinal fluid, also taken immediately upon death, revealed the presence of an organism of the Friedländer-aerogenes group and a paracolon bacillus. Autopsy revealed intraventricular hemorrhage, subdural and extradural hemorrrhages, and bilateral atelectasis.

Case No. 10. This patient was a full term infant who developed signs of intestinal obstruction forty hours after birth. Laparotomy revealed an atresia of the colon requiring colostomy. During surgery the mesentery of the cecum was accident-

ally injured necessitating exteriorization with subsequent resection. A scalp abscess appeared and culture of the pus revealed an organism of the Friedländer-aerogenes group. Four blood cultures were positive for the same organism. The patient expired despite the use of penicillin, streptomycin, sulfadiazine, aureomycin, and chloramphenicol.

Five patients, in the newborn period or shortly thereafter, developed septicemia and 4 of these succumbed. Three of the 5 were premature infants. Of these only 1 survived while the other 2 died at two and a half and four days of age, 1 with signs and symptoms of respiratory difficulty, the other with an extensive intracranial hemorrhage. Of the other 2 patients who succumbed, 1 was seriously debilitated by a severe pre-existing diarrhea and the other by surgical complications of the repair of atresia of the colon. The latter had pathologic evidence of meningitis at autopsy. It would appear from this very small series that Friedländer-aerogenes septicemia is more prone to occur in those infants who are debilitated by disease or the handicaps of prematurity. The sole survivor among the 5 appeared well at the time of the onset of the infection. The 3 patients who did not succumb at once developed jaundice.

Positive antemortem blood cultures were obtained in 2 patients. In a third an antemortem throat culture revealed the organism but a blood culture was negative. However, cultures of the blood and spinal fluid immediately upon death were positive. In the remaining 2 patients no antemortem cultures were carried out but the organism was recovered from the spinal fluid in 1 and from the blood and spinal fluid of the other immediately upon death. The death of these 2 infants arose primarily from the complications of prematurity. Baehr and his colleagues[2] contend that postmortem cultures which reveal the Friedländer bacillus are not entirely valid diagnostic criteria because of the tendency to preagonal invasion and postmortem contamination. On the other hand, Perlman and Bullowa[19] never found the organism in hundreds of preagonal and postmortem cultures in non-Friedländer cases. We are of the opinion that all 5 patients represent instances of Friedländer-aerogenes septicemia, albeit the clinical picture was dominated, in some, by other conditions.

THERAPY

Early trials with serum therapy for Friedländer's pneumonia yielded discouraging results.[19] Of the sulfonamide drugs, only sulfadiazine[20] appears to have any therapeutic effect experimentally and clinically, although one report found it without benefit in 6 of 7[21] patients with pneumonia. In our group, 1 patient with pneumonia made a prompt recovery on sulfadiazine and the patient with pyelonephritis presented evidence of improvement in his urinary infection before death.

Penicillin is, as a rule, of no value in these infections. Of the newer antibiotics, streptomycin,[22] aureomycin,[23] and chloramphenicol[23] are effective. So far as therapy is concerned, it is suggested that on the isolation of an organism of the Friedländer-aerogenes group streptomycin be administered. Sensitivity studies should be performed to determine whether aureomycin or chloramphenicol would be more effective.

SUMMARY

Because infections caused by the Friedländer-aerogenes group of organism are infrequent in children, we have recorded 10 cases, all infants, in which these organisms were encountered as pathogens. Because both *Aerobacter aerogenes* and Friedländer bacillus have similar morphologic, cultural, biochemical, and serologic characteristics, and because no valid criteria for their differentiation have been established, we are considering them as a single group. Diseases most commonly caused by these organisms are pneumonia, genito-urinary infections, meningitis, and septicemia and we have presented examples of each.

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TINEA CAPITIS; A SURVEY

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AND

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NEW ORLEANS

Although there has been a profusion of papers on the subject of tinea capitis in recent years, there have been no reports from this area. In a recent survey of colored dermatoses by Henington et al, from Charity Hospital in 1949, it was found that tinea capitis occurred in 212 out of 5,124 cases, or 9.56 per cent. In view of this incidence we feel that a survey of the type of organism in relation to race, sex and clinical picture is justified.

Tinea capitis is a superficial fungus infection of the scalp appearing predominantly in children before the age of puberty. It is extremely rare for this condition to appear for the first time in adult life. Clinically there appears a patchy alopecia with loosening and breaking off of the infected hairs. The hair loses its lustre and is easily removed from the infected follicle. There is associated a varying amount of fine branny scaliness in the area of alopecia; occasionally, an inflammatory process which varies from a few pustules to a painful, boggy erythematous localized tumefaction.

ETIOLOGY

The two most common organisms found are *Microsporon audouini* and *Microsporon lanosum*. *Microsporon audouini*, or the human form, is the predominant organism found in epidemics. *Microsporon lanosum* is so named for the characteristic yellow color found in cultures; this is synonymous with *Microsporon canis* and *Microsporon felineum*, so named after the animals which transmit the disease, namely, dogs and cats.

Tinea capitis may also be caused by *Microsporon fulvum*, *Trichophyton violaceum*, *Trichophyton crateriforme*, *Trichophyton sulfureum*, *Achorion schoenleini* (favus) and *Trichophyton gypseum*.

DIAGNOSIS

Diagnosis is made by one or more of the following methods: (1) Clinical appearance, (2) Wood light, (3) potassium hydroxide preparation of the hair, (4) culture on Sabouraud's or Litman's media.

Any circumscribed patch of broken-off hair seen in a child's scalp before puberty should be suspected as being tinea capitis.

The Wood light consists of any means of generating ultra-violet irradiation of approximately 3600A screened through a nickel cobalt filter. When the common Microsporon is the offender, the infected hair appears as luminous yellowish green stubs; in contrast with the Trichophytons

(gypseum and crateriforme) the infected hair appears dull bluish.

The microscopic examination consists of placing a few infected hairs in a drop or two of 10 per cent potassium hydroxide. Microscopically, there appears a mosaic sheath around the hair shaft.

Cultures should be done routinely and consist of implanting some infected hairs on a Sabouraud or Littman agar slant. The organism can be recognized readily by the specific gross and microscopic characteristics.

SURVEY AT CHARITY HOSPITAL

This survey consists of 134 cases of tinea capitis which were cultured for fungus between the last part of 1946 and through 1949 at Charity Hospital.

The age incidence varied from one to fourteen years. The most common age incidence was six years. (see Fig. 1). One hundred and sixteen were male and 18 were female. There were 124 colored and 24 white. Forty-five cases were epilated by the radiology department, all of which were caused by *Microsporon audouini*. Of those cases which were not epilated the duration of cure was one to eight months with an average of two months. Kerion was found in 12 cases of *Microsporon audouini* and 1 case of *Microsporon canis*. Of the 7 cases due to *Microsporon canis* 5 were white female; 1 in a colored female and 1 in a colored male.

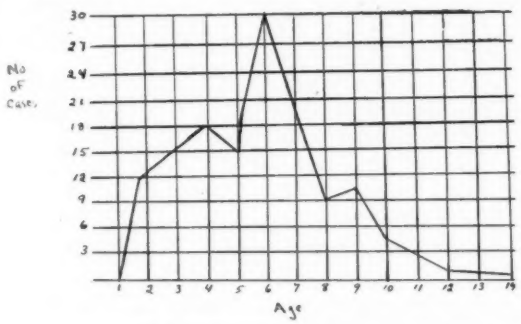

Fig. 1. Incidence of Tinea capitis according to age groups.

COMPARISON OF INCIDENCE

We have endeavored to compare our incidence with that of previous authors in table 1. Our findings conform with those of White (Boston), Livingsgood (Philadelphia), Bingeley (Cleveland), Benedict (Chicago), Lewis and Hopper. (1943-48).

In the following the predominant organism was *Microsporon lanosum*: Greenwood (Boston), Lewis and Hopper (N. Y. 1935-38), Weidman (Philadelphia), Pardo-Costello (Havana), Smith (El Paso), Shaw (Tenn.), Swartz (Boston).

Our interest in this subject started in 1946 when we cultured a number of cases of tinea capitis and found the predominant organism to be *Microsporon canis*. Since 1947, most of the cases of tinea capitis were routinely cultured by the pathology department of Charity Hospital and an analysis of the relationship of the prevalent

TABLE 1

REPORTED INCIDENCE

Author	No. Cases	Location	Year	M. Audouini	M. Canis	Others
White	279	Boston	1899	Aud. predominant		
Greenwood	40	Boston	1923	25 %	67.5%	7.5%
Lewis & Hopper	292	New York	1935-38	39.3%	39.6%	21 %
Weidman	36	Philadelphia	1941	41.7%	50 %	8.3%
Livingood	130	Philadelphia	1941	96.2%	3.1%	.7%
Pardo-Costello	32	Havana			100 %	
Binkley		Cleveland		70 %	30 %	
Smith		El Paso		rare	predominant	
Shaw	22	Tennessee	1942-46	0	95 %	5 %
Benedict	140	Chicago	1944	81.5%	12 %	
Dobes		Atlanta	1947	78 %	18 %	4 %
Snow	60	Birmingham	1948	78 %		
Swartz	256	Boston 1	1949	25.4%	65.3%	3 %
G. & G.	134	New Orleans	1946-49	82.8%	5.3%	11.9%

organism to year in which cultured appears in Table 2. From this table it can be

TABLE 2
PREVALENT ORGANISM

	M. Aud.	M. Canis	Others
1946	2	0	0
1947 (first half)	33	0	2
1947 (second half)	10	0	1
1948 (first half)	27	0	1
1948 (second half)	13	2	2
1949	26	5	4
	111	7	10

seen that there is some tendency towards an increase in *Microsporon canis* since the early part of 1948.

SUMMARY

We have reviewed 134 cases of ringworm of the scalp cultured at Charity Hospital from 1946 through 1949.

1. *Microsporon audouini* was found to be the predominant organism. (82.8 per cent.)

2. *Microsporon gypseum* was found in 3 cases; *Trichophyton gypseum* in 2 cases; and *Trichophyton sulfureum* in 2 cases.

3. Most common age incidence was 6 years.

4. Sex incidence was male predominating 6 to 1.

5. There were 124 colored and 10 white.

6. Clinically 16 cases showed inflammatory processes (kerion) of various degrees, of these 12 were due to *Microsporon audouini* and only 1 due to *Microsporon canis*.

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INFANTILE ECZEMA*

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NEW ORLEANS

In a review of 304 consecutive cases under 12 years of age observed during a period of one year, 21 per cent were allergic in origin; 18 per cent bacterial infections of the skin; 13 per cent hemangiomata; 12 per cent fungus infections; 10 per cent verrucae vulgaris; 8 per cent parasitic infections; and the rest, miscellaneous.

There were six types of allergic dermatosis in children. Of these, atopic dermatosis, otherwise known as infantile eczema, was by far the most frequent. Contact dermatitis, or dermatitis venenata, was second in order of frequency. Only rarely did we see lichen urticatus, urticaria pigmentosa, drug allergy, or urticaria itself. I shall limit my paper to a discussion of infantile eczema.

Let us first define infantile eczema. It is a dermatitis which occurs in infants and young children, usually before six months of age, and nearly always before the age of two years. It is one of the group of atopic diseases occurring only in human beings, and associated with the production of circulating antigens. Other members of this group include asthma, hay fever, and hives. The shock tissue is the subepidermal vascu-

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lar network, and the rash results from the formation of minute vesicles in this level of the skin. They soon rupture onto the surface, producing rough red patches, pea-sized or larger, accompanied by extensive itching and excoriations. The initial site, characteristically, is the cheeks.

The family history in these cases is predominantly allergic. There is also a decided seasonal variation, the disease being limited almost exclusively to the colder months.

The precipitating etiological factor in early infancy is often the diet. The affected babies are usually on a total or supplementary diet of cow's milk. In fact, the incidence of infantile eczema is seven times as great in those who are on a total diet of cow's milk as it is in those fed exclusively on breast milk.[1] It is three times as great in those on a mixture of cow's milk and breast milk. We often get a history of diarrhea and vomiting associated with milk, orange juice, egg, or some other food. Inhalants, as an etiological factor, play an insignificant part before the age of two years, but increase in importance thereafter.

Contact dermatitis is often a complicating factor in these cases. Such children are frequently subject to contact irritation such as rubber or plastic plants, and baby oil containing hydraquinone. In treatment, we must prohibit the use of these substances. Wool is always an irritant, and we substitute linen, cotton, or other materials whenever possible.

The cutaneous wheal test is of no etiological significance in these cases. Wheal test-positive substances will not produce a dermatitis upon exposure to these same substances during dermatitis-free periods.[2] Patch tests are of value in epidermal sensitivity when contact allergy is also present, that is, skin sensitivity to rubber pants, oils, and other irritants.

The principal complications are:

1. Secondary pyogenic infection, with a considerable degree of pustulation and crusting.

2. Kaposi's varicelliform eruption, due to the herpes simplex virus which produces a herpetic type of eruption occurring in crops. This was often fatal before the advent of aureomycin.

3. Generalized vaccinia, due to infection with the smallpox virus.

MANAGEMENT

Preventive: We have previously stressed the high incidence of this condition in children with allergic backgrounds. It is of the utmost importance that these children be given breast milk, since it has been shown that the incidence of infantile eczema is seven times greater with any other milk.

Treatment: A child who has already developed infantile eczema should be fed canned goat's milk. All other foods should be temporarily discontinued, such as orange juice, tomato juice, and egg; and vitamin concentrates should be substituted. Later on, pureed fruits may be added, and from time to time meats and vegetables.

Bathing care consists of oatmeal-soda baths once a day, omitting the use of soap.

Diaper care—We substitute mineral oil for baby oils in cleaning after a stool. The diapers should be rinsed in the toilet and soaked in boric acid solution before washing. Rubber or plastic pants are contraindicated.

Topical medication—With secondary infections aureomycin or bacitracin ointment is useful. In the dry stage, crude coal tar 1 per cent in Lassar's paste.

Oral medication—Antihistaminic drugs give symptomatic relief and induce rest.

Clothing—Avoid wool in contact with the skin.

Restraint—This is harmful to both child and parents and should not be resorted to. White cotton socks drawn over the hands and pinned to the gown afford sufficient protection.

Immunizations—These should be given in summer, when the incidence of the disease is low. Special antitoxins are available for patients with high sensitivities.

In general, the parents should be warned that recurrences should be expected, especially with every drop in temperature,

and that the future may hold for the patient this and other types of allergy.

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DISCUSSION

Dr. W. C. Rivenbark, (New Orleans): Allow me to compliment Dr. Kennedy on his very excellent and concise presentation of the subject of infantile eczema. A few additional remarks are fitting at this time because of the stubborness of this condition in responding to present therapy. These patients often migrate in a circle from pediatrician to dermatologist to allergist.

This condition has very marked cosmetic angles because the mother resents having relatives, friends, and neighbors see her baby with such an awful looking face. I would like to stress the importance of informing these patients from the beginning that:

1. Your baby probably will get well; most do.
2. The length of time to effect a cure is very variable and definitely not rapid.
3. They must have patience and follow specific instructions of therapy, each factor of treatment being as important as the other.

Dr. Kennedy has pointed out that this condition was the most frequently seen of all skin diseases in infants and children under 12 years of age during his survey. It is also noted seven times more frequently in artificially fed infants. This fact is easy to explain because as of today, in spite of all our efforts to promote breast feeding, less than 1 out of 20 mothers nurse their babies.

I agree with Dr. Kennedy that wheal test positive substances will not in a great majority of patients produce a dermatitis upon exposure. I have seen children who had been placed on such radical elimination diets that the resulting nutritional disturbance was worse than the eczema, and the eczema had not improved.

Treatment: 1. Goats milk, mullsoy, or hypoallergic milks must be used.

2. Substitutes for orange juice are necessary in a great number of cases in the form of vitamin C concentrates. Other vitamins should be given in adequate dosage.

3. Other foods—We must not overlook the fact that fast-growing infants and children need all of the essential foods. Unless one is absolutely sure that the condition is due to one or more specific essential foods, they must not be eliminated, in order to prevent a severe nutritional disturbance.

4. Topical medication: It is generally agreed that crude coal tar preparations are the most effective, however, I personally like to use resorcin, 20 grains to the ounce in vaseline, as well as tar preparations.

5. Restraint: I am sure that trauma is harmful in this condition, therefore, we must restrain to prevent scratching, by use of socks pinned to the gown, mechanical arm splints such as cardboard, tongue blades and Prevent-a-habits (made of wire).

6. Immunizations: I have always been successful in immunizing these infants and children without increasing the manifestations of the disease.

7. Recently, some investigators have treated infantile eczema with small intradermal doses of staphylococcus toxoid. They report the results as being quite favorable.

8. Use of essential unsaturated fatty acids: Hansen, University of Texas, has shown that there is a relation between disturbance in essential fatty acid metabolism and the occurrence of infantile eczema. The specific role of the essential fatty acids appears to be that of restoring and maintaining normal nutrition of the skin. He experimented with lard, corn oil, linseed oil, olive oil, and tar, and found that lard was the most beneficial as a supplement to the diet.

Fairly recently, Sharp and Dohme have developed a product containing the essential unsaturated fatty acids present in vegetable oil combined with vitamin A and wheat germ oil for the treatment of this condition. I personally have used this on numerous cases and believe it to be beneficial. The name of the preparation is Esselip.

It is a fact that cow's milk is low in essential fatty acids, therefore the increased incidence of the disease in artificially fed infants. Hansen concluded that the diet must include fats rich in unsaturated fatty acids, and was of definite benefit in a majority of eczematous patients. It is further emphasized that this is only one factor in the treatment, and alone will not cure the disease.

Conclusion: Meticulous investigation and elimination from the diet of foods to which the patient is positively known to be sensitive; administration of substances known to be high in essential unsaturated fatty acids; topical applications of crude coal tar; elimination of irritating factors; and maintenance of adequate diet for normal nutrition are all important factors.

ROCKY MOUNTAIN SPOTTED FEVER

REPORT OF 2 CASES IN LOUISIANA

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AND

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NEW ORLEANS

Rocky Mountain spotted fever (variously called tick-bite fever, spotted fever, and black fever), is a tick-borne rickettsial disease which has been reported from 43 of the United States as well as from Canada and several Latin American countries. Since the mortality in untreated cases ranges from 10 to 80 per cent, depending on the age of the infected persons and possibly on the virulence of the local strain of rickettsia in the tick population, the early recognition of the disease and its proper management are matters of considerable importance to the practicing physician.

During the month of June 1950, two children from different localities in Tangipahoa parish, Louisiana, were referred to the Charity Hospital at New Orleans with a febrile disease later shown to be Rocky Mountain spotted fever. The simultaneous occurrence of two cases of this disease, previously rather uncommon in Louisiana, together with some interesting features of their clinical course and therapy, prompted the present report.

CASE REPORTS

Case No. 1. On June 1, 1950, L. Y., a 2½ year old white male child, was admitted to the Independent Pediatric Service of Charity Hospital because of fever and rash. His illness began May 21, 1950, when he became lethargic, developed fever, and complained of sore throat. On May 23, 1950, he was seen by a private physician who prescribed penicillin and aspirin to be taken orally every two hours. Several hours later the parents noted that the child had developed a rash. This rash appeared first on the extremities and spread centripetally, rapidly covering his entire body; it was described as resembling the rash of measles.

The patient failed to improve and on May 26 and 27 he was given penicillin intramuscularly. Sulfonamide was begun orally on May 26 and aspirin was administered daily. He seemed to improve on May 29 and 30 with decrease in fever, lethargy, and rash; but his condition again became worse on June 1, and he was admitted to the Charity Hospital.

The child lived on a farm near Wadesboro, Louisiana, and the parents stated that numerous ticks were present in the community. A live tick had been removed from the patient's scalp two days prior to the onset of his illness.

Physical examination: Examination made on admission revealed a two year old white male child, weighing 26 lbs., who was acutely ill and quite lethargic. His rectal temperature was 103.6°F. A discrete, rose, maculopapular rash was present. The rash was generalized with some accentuation over the extremities, covering the palms of his hands and the soles of his feet. There was bilateral periorbital edema, also nonpitting edema of the dorsum of the hands and feet. The breath sounds were diminished over the right mid lung field posteriorly. A moderately loud blowing systolic murmur was heard over the precordium and the heart sounds were of poor quality. The liver was felt 3 fingerbreadths below the costal margin and the spleen was palpable. General glandular enlargement was present.

Labaratory data on admission: The hemoglobin was 10 gm., the red blood cell count 3,200,000, the erythrocyte sedimentation rate 6 mm. (corrected), the coagulation time 3 minutes. The white blood cell count was 8,200 with 82 per cent neutrophils and 18 per cent lymphocytes. Urinalysis revealed a trace of albumin only. Serologic test for syphilis and the Mantoux were both negative. Blood culture yielded a saprophytic staphylococcus, presumably a contaminant. Chest x-ray showed right hilar infiltration.

Course in hospital: A tentative diagnosis of Rocky Mountain spotted fever was made and oral aureomycin was begun in a dose of 100 mgm. every six hours. On June 2, because of vomiting, intravenous administration of the drug became necessary; the dose was 20 mg. every six hours for three days. On the same day the rash, previously macular, became more hemorrhagic and in numerous areas appeared confluent. Clinical signs of cardiac failure appeared, such as basal pulmonary congestion and a further increase in liver size, and the electrocardiogram showed prolongation of the PR interval (0.24 seconds) and right axis deviation. The patient was therefore given oxygen, digitoxin, and mercuhydrin.

From the fourth hospital day on, the hospital course was characterized by gradual lysis of fever with return to normal temperature on the eighth

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hospital day. There was no increase in the rash after intravenous aureomycin was begun, and it began to fade gradually on the fourth hospital day. The heart returned to normal, and the liver and spleen were no longer palpably enlarged after June 6. The edema disappeared at about the same time. Supportive therapy consisted of two transfusions and daily intravenous fluids during the first five days of hospitalization. Oral aureomycin was given from June 5 on, and was discontinued entirely on June 14.

The patient was discharged clinically well on June 16, 1950, the only residual evidence of his disease being a few fading cutaneous hemorrhagic lesions.

The results of serologic studies are shown in Table 1.

TABLE 1

RESULTS OF SEROLOGIC TESTS MADE IN TWO CASES OF ROCKY MOUNTAIN SPOTTED FEVER

Case No.	Date of Serum Specimen	Type of Test	Antigen Employed	Titer Observed
1	6/ 2/50	Aggl.	OX19	1:160
	6/ 6/50	Aggl.	OX19	1:2560
	6/12/50	Aggl.	OX19	1:1280
	6/ 2/50	complement fixation	RMSF murine typhus Rickettsial-pox "Q" fever	all negative
	7/ 7/50	complement fixation	RMSF	1:128
2	6/ 6/50*	Aggl.	OX19	negative
	6/ 9/50	Aggl.	OX19	negative
	6/13/50*	Aggl.	OX19	1:40
	6/16/50*	Aggl.	OX19	1:40
	6/19/50	Aggl.	OX19	negative
	6/ 6/50**	complement fixation	RMSF	negative
	6/13/50**	complement fixation	RMSF	1:8192**
	6/19/50	complement fixation	RMSF	1:8192**

*agglutination tests with typhoid O and H, paratyphoid, tularemia and brucella antigens were all negative.

**complement fixation tests with other rickettsial antigens were all negative.

Case No. 2. On June 4, 1950, H. M., an 11 year old white girl, was admitted to the Tulane Pediatric Service of Charity Hospital with a history of illness of seven days' duration. She first complained of sore throat and refused her food. On the following day she developed fever, and on the third day a transient rash was noted on her legs. On the fifth day fever and anorexia persisted and a physician was consulted. The child's temperature at that time was 105°F. and a purpuric rash was present over the face, abdomen, and extremities. The physician gave her 300,000 units of procaine penicillin G with 100,000 units of crystalline penicillin in an aqueous dispersion, and prescribed 0.5 gm. of sulfadiazine every four hours. The rash was more intense on the following day and the child's temperature was 104.5°F. Another dose of penicillin (400,000 units) was given, also 0.5 gm. of dihydrostreptomycin; the sulfadiazine, however, was discontinued. On the seventh day of her illness she again received penicillin and dihydrostreptomycin, as well as a dose of aureomycin. Despite these measures the patient failed to improve and was referred to the hospital for admission.

Upon further questioning it was learned that the patient lived on a farm in Tangipahoa parish, and had frequently found ticks on herself. The mother recalled removing a tick from the patient four days before the onset of the present illness.

Physical examination: Examination made on admission disclosed a dangerously ill, moderately dehydrated girl of about 11 years of age. She responded when spoken to, but for the most part talked incoherently. She was covered by a blotchy purpuric rash which was most intense over the extremities. Her temperature was 105°F., yet her extremities felt cold to the touch. Pulse and blood pressure were not obtainable, and the patient's nail beds and lips were cyanotic. Breathing was somewhat labored, but there were no rales heard over the lung fields. The heart tones were of good quality and the apical rate 160 per minute. The liver and spleen were not palpable. The deep reflexes were hypoactive, although the back and neck were somewhat resistant to flexion.

Laboratory data on admission: The hemoglobin was 14.5 gm., the white blood cell count 3,400 with 37 per cent neutrophils (26 per cent band forms), 2 per cent eosinophils, 9 per cent monocytes and 52 per cent lymphocytes. The blood culture yielded no growth, and stained smears of the petechiae revealed no bacteria. The tourniquet test was strongly positive. Urinalysis revealed 2 plus albumin and an occasional red blood cell. The spinal fluid was clear, with a pressure of 130 mm.; it contained 8 lymphocytes per cubic mm., 18 mgm. per cent of protein and 76 mgm. per cent of glucose, and was sterile on culture. Serologic test for syphilis and the Mantoux were negative. An

electrocardiogram showed prolongation of the PR interval and was interpreted as showing evidence of myocardial disease.

Course in hospital: Measures were immediately instituted to combat the patient's shock-like state: An infusion of glucose, plasma, and electrolyte solutions was started at once and the patient was placed in an oxygen tent. She also received a transfusion of whole blood, and 50 cc. of aqueous adrenal cortical extract intravenously. Her blood pressure, previously unobtainable, rose to 116/70. She was also given a single dose consisting of 1.5 gm. of sodium sulfadiazine intravenously, and was started on 200,000 units of aqueous penicillin every two hours, as well as 250 mg. of aureomycin by stomach tube every six hours.

Because of the desperate condition of the patient on admission, the initial therapy was started before an adequate history could be taken. A more complete history made the diagnosis of Rocky Mountain spotted fever the most likely one, so the sulfadiazine was discontinued after one dose.

A constant infusion of glucose, plasma and electrolyte solutions was maintained for the first four hospital days in order to stabilize the blood pressure. Twenty cc. of adrenal cortical extract were added to each 1000 cc. of fluid administered. Blood pressure readings were taken every fifteen minutes and an additional 10cc. of adrenal cortical extract were given intravenously each time the blood pressure fell below a systolic reading of 85 mm. of mercury. Cyanosis was present for about twelve hours after admission, then cleared. Generalized edema developed on the second hospital day, but gradually disappeared. Urinary output was satisfactory but it was necessary to catheterize the child at intervals during the first three days of treatment. There was a bloody discharge from the rectum and the vagina as well as slight oral bleeding during the first three hospital days. By the second hospital day she tolerated sips of water and from the fourth hospital day on she was able to take an adequate soft diet. By this time the blood pressure was well stabilized and no further adrenal cortical extract was necessary. The temperature fluctuated between 100.4°F. and 105°F. for the first seven hospital days and then dropped gradually, reaching normal on the thirteenth hospital day. The skin rash, at first purplish in color, gradually turned brown. A few small areas became necrotic but most of the lesions dried up. All medications were discontinued on the sixteenth day at which time the patient appeared quite well, though still very weak.

The results of the serologic examinations made are presented in Table 1.

TABLE 2

CASES OF ROCKY MOUNTAIN SPOTTED FEVER REPORTED IN LOUISIANA DURING THE YEARS 1940-1949[4]

YEAR	CASES	PARISH
1940	0	
1941	46 yr. W.M.	Rapides
1942	14 yr. W.M.	Caddo
1943	28 yr. W.M.*	Vernon
	F (age, race?)*	Winn
1944	10 yr. C.M.*	Caddo
	49 yr. W.M.*	Winn
	50 yr. W.M.	West Feliciana
1945	11 yr. W.M.	Vernon
1946	37 yr. W.M.	Union
	7 yr. W.M.*	Webster
	32 yr. W.M.	Tangipahoa
	14 yr. W.M.	Tangipahoa
1947	7 yr. W.F.	Tangipahoa
	12 yr. C.M.	Caddo
	12 yr. W.M.	Rapides
1948	36 yr. W.M.*	Tangipahoa
	14 yr. C.M.*	La Salle
	57 yr. W.F.	Webster
	52 yr. W.M.*	Orleans
1949	W.M. (age?)	Caddo

*Known deaths. Other deaths may have occurred, but are not so listed in the files of the Health Department.

DISCUSSION

Epidemiology in Louisiana. The incidence and geographic distribution of cases of tick-bite fever in Louisiana can be seen at a glance from Table 2.

Ticks which have been incriminated in the transmission of Rocky Mountain spotted fever in this geographic area are principally *Dermatocenter variabilis* (the American dog tick) and *Amblyomma americanum* (the Lone Star tick.) However, two other local species, *Rhipicephalus sanguineus* (the brown dog tick) and *Amblyomma maculatum* have been proven experimentally to be capable vectors of rickettsiae.[1, 9]

Dogs, other domestic animals, certain birds, and a number of small wild animals

are the usual hosts of the tick vectors.[3] These hosts not only afford vital nourishment for the ticks but also, in many cases, undergo an active though usually silent infection during the course of which they may spread the agent, *Dermocentroxenus rickettsii*, to new individual vectors. Ticks may become infected at any stage of development, and, once infected they remain so for life; furthermore, they may pass the agent through the ovum to succeeding generations of ticks. Man, in tick infested areas, becomes infected through careless exposure to tick bites, or less commonly, through contamination from manual removal of ticks which are feeding on domestic animals.

Clinical picture. The severity of the disease is quite variable, some individuals suffering only mild abortive attacks which do not confine them to bed, while in other cases the infection runs a rapidly fatal course.[3] The incubation period may vary from two to twelve days, averaging six to seven days. The onset is sometimes gradual, marked by anorexia and malaise, as in the second patient patient reported here; again it may be abrupt, with chills and fever. The fever usually climbs for a week or ten days, frequently with a morning remission, remains high for another week or so, and falls rather suddenly in the third week. The rash often appears early as a transient mottling, as described in our second patient, soon afterwards as light red macules on the wrists and ankles. It spreads centripetally to the proximal portions of the extremities, the face and the trunk; and characteristically involves also the palms and soles. Within a short time the lesions usually become petechial; in severe cases only are there large violaceous areas with subsequent necrosis. Patients with extensive purpuric rashes usually succumb early in the disease to circulatory collapse; whereas those who survive until the second week are more apt to suffer other complications, such as myocarditis, encephalitis and pneumonia. The clinical features of the disease can all be related to the underlying pathologic lesions of the capillary blood vessels, which consist of areas of proliferation in the endothelium, followed by thrombosis and degeneration.[8] Widespread capillary damage is responsible not only for small areas of hemorrhage in the skin, brain, heart, spleen, liver, and genital organs, but probably also for the generalized edema which is so often present, and which was a striking feature in our two patients. Capillary damage is also responsible for the occasional occurrence of peripheral circulatory failure,[6] which was prominent in our second case.

Diagnosis. Meningococcemia, typhoid fever, measles, and murine typhus are the diseases most likely to be confused with tick-bite fever, even by experienced observers. The slow onset in our second patient, the failure to respond promptly to sulfonamides, the absence of meningococci in the petechial smears as well as in blood cultures, and the normal spinal fluid, pointed towards an infection with some agent other than the meningococcus, whereas murine typhus seemed unlikely because of the severity of the symptoms. In the first patient the peripheral distribution of the rash, the generalized edema and the presence of moderately severe myocarditis all seemed more characteristic of tick-bite fever than of murine typhus.

In final analysis, however, the clinical diagnosis must be confirmed by serologic tests, of which two are widely used: the Weil-Felix reaction and the complement fixation test.[3] For both tests serum is obtained from the patient as soon as the diagnosis is suspected, and again during convalescence in order to detect a rise in antibody titer. In the Weil-Felix reaction, the patient's serum is tested for agglutinins against Proteus OX19. While a titer of 1:320 is considered to be of diagnostic significance, not all patients achieve such a titer and a few fail to produce agglutinins at all, or produce them only late in convalescence. Furthermore, the development of a positive Weil-Felix reaction is commonly observed also in typhus fever and occasionally in various nonrickettsial infections. The complement fixation test presents two

advantages over the Weil-Felix reaction: (1) It is highly specific and enables accurate differentiation between Rocky Mountain spotted fever and other rickettsial diseases such as murine typhus and rickettsialpox; and (2) it remains positive for many years after infection.

The serologic studies made in the present cases illustrate some of the difficulties which may be encountered. In Case No. 1 the clinical diagnosis received early and strong support in the rapid rise of the OX19 agglutinins to a significantly high titer (1:2560). The delay in manifestation of the complement fixing antibodies, however, remains unexplained. In Case No. 2 the OX19 agglutinins appeared late, only transiently, and never in diagnostic titer. The complement fixing antibodies, on the other hand, appeared promptly and in high titer, thus confirming the diagnosis.

Treatment. Sulfonamides were tried in the treatment of tick-bite fever,[13] but it was early shown that administration of sulfonamides was probably actually harmful. Sulfonamide treated guinea pigs died sooner than did the untreated controls. It was, in fact, the deleterious effect of sulfonamides in rickettsial infections which led to a trial of the metabolic antagonist, para-aminobenzoic acid (PABA), as a therapeutic agent first in experimental animals and later in humans.[11, 14] In both of the cases presented here, the patient's condition appeared to deteriorate soon after sulfadiazine therapy was instituted. Since sulfonamides are the most effective therapeutic agent known at present for meningococcal infections, the importance of early differentiation between meningococcemia and tick-bite fever can readily be appreciated.

While PABA was extremely effective in reducing the mortality from tick-bite fever, it was not without toxic effects; also the results were disappointing in patients treated later than the eighth day from onset of symptoms.[11, 14]. Aureomycin and chloramphenicol, on the other hand, have given excellent results, regardless of the duration of symptoms prior to therapy.[2, 7, 10, 12] Pincoffs[10] and associates found the average duration of fever after the start of chloramphenicol therapy to be 2.2 days. In our first case the temperature fell dramatically. The second patient showed prompt clinical improvement, and the persistence of low grade fever over a seven day period may well have been due to the extensive tissue necrosis at the sites of hemorrhage. Although aureomycin was employed in both of our cases, available experience suggests that there is little to choose between it and chloramphenicol, both agents being equally effective.

Peripheral circulatory collapse, when it occurs, is rapidly fatal unless energetically treated.[5] The need for intravenous plasma, whole blood, glucose, electrolytes, and vitamins cannot be overemphasized. We believe, on the basis of favorable experience in the use of adrenal cortical extract in peripheral circulatory collapse with meningococcemia, that this substance should be used in tick-bite fever also, whenever the blood pressure is low or unobtainable.

SUMMARY

1. Two cases of Rocky Mountain spotted fever in children are presented.

2. The epidemiology of tick-bite fever in Louisiana is discussed.

3. The clinical manifestations of the disease are briefly reviewed, with emphasis on differential diagnosis.

4. Various aspects of chemotherapy are briefly discussed, with a warning against the administration of sulfonamides to patients with possible tick-bite fever.

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PRACTICAL TREATMENT OF THE DIARRHEAS OF EARLY LIFE

EDWIN A. SOCOLA, M. D.

NEW ORLEANS

Although more scientific methods have replaced the time-worn antiquated treatment of diarrhea with barley water and bismuth, the mortality from the diarrheal diseases in early life remains relatively high in some sections of this country. Because of the fact that by better nutrition of infants and higher standards of general hygiene and sanitation, the deaths from diarrhea can be reduced, the practitioner should feel his responsibility in his community in educating the public to better nutritional care of the well baby and in obtaining satisfactory local sanitary conditions.

CLASSIFICATION

The diarrheas of infancy and childhood can be divided into the following groups:

1. Simple (Noninfectious, dietetic)
2. Parenteral
3. Infectious
   a. Bacterial
   b. Protozoal
   c. Virus
4. Diseases associated with diarrhea (celiac disease)
5. Miscellaneous (nervous, allergic)

The simple diarrheas are those diarrheas caused by a dietary indiscretion (mechanical) or the inability of the infant to digest some part of the diet (fermentative) or carbohydrate, and putrefactive, or protein diarrheas.

The parenteral diarrheas are those caused by an infection outside of the digestive tract such as otitis media, urinary infections, and pneumonitis.

The infectious diarrheas present an infection in the digestive tract caused by (1) bacteria such as strains of *B. dysenteriae*, or salmonella; (2) *Endameba histolytica* and other protozoal incitants; (3) or of virus origin.

There are some diseases in which diarrhea is the most prominent and outstanding symptom, such as celiac disease.

The miscellaneous diarrheas include all of those that do not fall into the previously mentioned groups, such as the allergic and nervous diarrheas.

THERAPY

Up to a few years ago, one of the primary objects of treatment was to alter the bacterial flora, i.e., the feeding of protein food to correct a fermentative diarrhea, or the intake of carbohydrate food in a putrefactive diarrhea. In fact, twenty years ago many practitioners carried litmus paper in their bags to determine the reaction of the stools.

At the present time, therapy resolves itself into the prevention, the control, or the correction of dehydration. Darrow[1] emphasizes that practically no deaths occur from diarrhea without the development of dehydration, and that by the replacement of water and electrolyte losses the mortality figures can be kept very low.

Correction of the dehydration and the acid base equilibrium in the severe diarrheas is accomplished by intravenous administration of fluids. More or less standardized methods are employed in various pediatric centers. With the possible exception of water, glucose, and salt solutions, it

Presented at the Seventieth Annual Meeting of the Louisiana State Medical Society, April 26, 1950, in Baton Rouge.

is the common procedure in many institutions to withhold all fluids by mouth during the period of active diarrhea and parenteral administration of fluid, hence the expression, "Splint the bowel." There are many pediatricians who argue that prolonged intravenous therapy has been carried too far and that the pendulum is beginning to swing to the other side, i.e., to earlier oral feeding. O'Keefe[2] feels that:

"The starvation treatment currently practiced in the diarrhea of infancy is certainly a factor in the production of ketosis and the depletion of water and minerals—conditions which one attempts to remedy by concomitant intravenous administration. Such therapy takes cognizance of the water and mineral requirements of the infant. The caloric and protein requirements are, however, usually ignored or at best unfulfilled. The consequent protein deficiency results in a depletion of antibodies and an inevitable deterioration in the resistance of the infant to infection. The aforementioned inadequate oral diet is open to the same criticism.

"In this disease, recovery depends to a great extent on maintenance of the natural defenses, namely, the antibodies. Such maintenance can be attained best and, perhaps, only by oral feeding of the sick infant."

For everyday working purposes, the clinical classification of the diarrheas is suitable for therapeutic purposes.

	Vomiting	Dehydration	Toxic symptoms	Blood CO_2
Mild	0 to ++	0	0	Normal
Moderately Severe	+ to +++	+ to ++	+ to ++	Above 30
Severe	++ to ++++	++++	++++	Under 30

The presence of vomiting with diarrhea can lead rapidly to dehydration and acidosis. As previously stated, the severity of a diarrhea can be measured usually by the amount of dehydration.

Toxic symptoms, such as hyperpyrexia, circulatory disturbances with anoxia of the tissues, meningismus, and shock, are met with in the severe diarrheas.

The determination of the carbon dioxide content of the blood should be considered as an emergency laboratory procedure. In a moderately severe or severe diarrhea by the Van Slyke manometric method, the pathologist can give a report in a few minutes. It is essential that the degree of acidosis, if present, be known in order to correct it with the proper intravenous solutions. In the handling of even the mildest of diarrheas, it must be remembered that a failure to correct the condition, especially if vomiting is present, can result in dehydration and acidosis.

For several years, the essayist has used the following modified Ringer's solution, with the addition of Coca-Cola orally, in the mild and moderately severe diarrheas accompanied by vomiting. Even in the treatment of the severe types, this mixture has proved of value combined with the parenteral administration of fluid, and chemotherapy.

Rx Sodii Chloridi gr. 108
Potassii Chloridi gr. 5
Calcii Chloridi gr. 4
Aqua Dest. enough to make 1 ounce

1. Dilute the contents of prescription bottle in 1-½ quarts of cool boiled water.
2. Keep in ice box.
3. Over six months of age—take 3 ounces of this dilution (cold) and 2 ounces of cold Coca Cola (60 per cent diluted Ringer's solution and 40 per cent Coca Cola) equals 5 ounces and offer every two hours day and night for twenty-four hours.
4. After vomiting has been arrested for over twenty-four hours, and the infant has retained this mixture, banana powder (meloripe) therapy may be started.
   Method:—One level tablespoonful of meloripe banana powder is dissolved in 1-½ to 2 ounces of water per pound of body weight in twenty-four hours. The total calculated amount of banana powder and water is divided into ten equal feedings and is offered at 6 A. M., 8, 10, 12, 2 P. M., 4, 6, 8, 10, 2 A. M. for at least forty-eight hours.

Before resuming the regular diet, a transition diet consisting of powdered or liquid skimmed milk, or a lactic acid skimmed milk, with meloripe as the added carbohydrate, is employed as the basic food for at least forty-eight hours. The pectin agar in dextri maltose formula as well as some of the relatively high carbohydrate,

low fat, and low protein proprietary infant foods may serve as suitable temporary foods during the transition period. In this transition diet, beef broth, dry toast, crackers, ripe bananas pressed through a strainer or creamed, may also be employed. A return to the regular diet is largely determined by the severity of the treated diarrhea but the transition period should be at least forty-eight hours.

The modified Ringer's solution and Coca-Cola mixture, if given cold, is readily retained by the infant often when glucose, water, crushed ice, etc., have been repeatedly regurgitated. Bradley[3] employs the Coca-Cola syrup in teaspoonful doses in the control of epidemic vomiting and feels that the spectacular results can perhaps be attributed to the xanthine content, which he thinks may reduce the gastric tone.

The essayist feels that a fluid that is retained orally and which does contain the chlorides of sodium, potassium, and calcium, even in weak concentrations, with the carbohydrate of Coca-Cola, and from clinical experience does not aggravate the diarrhea, is certainly indicated in the correction of dehydration and electrolyte loss in the diarrheas of moderate severity. Furthermore, in the severe diarrheas it can be used at the same time that the correction of the acid base equilibrium and dehydration is being carried out by intravenous therapy.

In the choice of banana powder, following the Ringer's solution—Coca-Cola mixture, the essayist feels that this food can safely be used and wishes to call attention to the relatively high potassium content of the banana. In this manner, the potassium intake is administered without the dangers of potassium poisoning.

In a previous study[4] of banana and banana powder therapy in the diarrheas of early life, the essayist was able to demonstrate an average gain in weight of 8.1 oz. after ninety-six hours of treatment in 15 cases of infectious diarrhea.

Briefly, it may be stated that of the many advances in the treatment of the severe diarrheas, the use of potassium as advocated by Darrow is an outstanding scientific accomplishment. The correction of moderate acidosis by sixth molar lactate solution and of severe acidosis by sodium bicarbonate solution, intravenously, is accepted in most centers. The proper use of glucose and normal saline solutions in combating dehydration constitutes part of the usual standard intravenous methods. Plasma and blood transfusions are used to overcome shock early and the latter is also used later for secondary anemia.

As to chemotherapy, penicillin may be administered intramuscularly but a great deal of care should be exercised in the use of sulfadiazine until the oliguria has been corrected. Sulfaguanidine has been used extensively. The newer drugs, aureomycin and chloromycetin, may prove of value in the diarrhea of virus etiology. Streptomycin, it is claimed, has helped some salmonella infections. Blood, irradiated by the Knott Hemo Irradiator, is now being tried also in the virus diarrheas.

Vitamin therapy is of greater value during the convalescent than in the brief acute stage.

Proper treatment of the condition causing a parenteral diarrhea is required before improvement can be expected.

Allergic diarrheas are being detected more and more at the present time. Recently, F. D. Nance of Honolulu introduced a method of demonstrating eosinophilia in the stools of allergic diarrhea. Treatment consists of detecting the causative allergens by skin tests or elimination diet. As cow's milk is often the etiological factor, a hypo allergic milk is ordered and later desensitization is carried out.

Diodoquin because of its low toxicity and high effectiveness is probably the drug of choice in the amebic dysentery of children. Recent reports of the use of aureomycin in this condition are very encouraging.

SUMMARY

A method is presented by which (1) the mild and most of the moderately severe diarrheas can be corrected without resorting to the parenteral administration of fluid. (2) Although intravenous therapy is indicated in some of the moderately severe

diarrheas and *all* of the severe diarrheas, a more rapid and satisfactory recovery results when early oral administration of a suitable corrective liquid and food is possible, such as with the one described in this paper.

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Medical and Surgical Journal

Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

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News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

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THE POSITION OF ORGANIZED MEDICINE SINCE THE CONGRESSIONAL ELECTIONS

Now that the Congressional elections are over, it is fitting that organized medicine should consider its position and formulate plans. Three facts emerge for consideration of the situation which are of importance to physicians.

The first of these concerns what appears to be a reasonable conclusion that there is a trend away from Communism and toward a sense of responsibility for the ultimate good of the community, as distinguished from the immediate welfare of the Party. This was responsible for whittling down the Democratic majority and putting the southern members of Congress in a position where their precarious influence has been turned to a real balance of power. With a small majority in the House of Representatives and a majority of only two in the Senate, it is almost inconceivable that the Democratic Party could enact any legislation definitely detrimental to the medical profession. Compulsory sickness insurance, in any guise, is postponed. This happy situation, while it has in it the elements of risk which may breed overconfidence, is nonetheless welcome, giving us a period of time in which we may formulate further plans. We should not look upon the situation as though an invader had been defeated and the army destroyed. We should, on the other hand, consider it as one in which a tactical victory had been obtained, but now, because of this victory, guerilla warfare must be fought and the obvious ill effects of infiltration must be neutralized.

The second fact of some importance and of much satisfaction to the doctors is that their influence was instrumental in contributing to the shift in public opinion, in some degree almost everywhere, and to a considerable degree in certain states. It is well to mention here the defeat of Senator Pepper in Florida. It is also reported that in recent weeks Oscar Ewing proposed that he campaign for the Democrats in his native state of Indiana. His offer was greeted with an enthusiastic "No" by his own party members, the inference being that they had trouble enough and did not wish to struggle any harder against medical opposition than they were doing already. These two observations are a compliment to the doctors and indicate an increased effectiveness of our influence as citizens. For too long a time, have we remained inactive, or possibly, aloof from exercising our proper influence in community, political, or economic organizations.

The third fact which emerges from among these considerations is that medicine, as an organization or as individuals, must utilize the next two years to consolidate the ground gained, and to extend our broad educational program. Each physi-

cian must feel that an important part of the public relations problem of his profession is in his hands. Local societies should continue to send to various civic bodies speakers on medical problems. Legislative committees should exact statements of policy from prospective candidates. Points of friction between the profession and the laity should be relieved. A greater sense of solidarity should be built up within our organization. In every instance the guiding star should be to do that which would promote better medicine. In this way, we will be able to restore the doctor to the position he occupied some years back, and to pass on the noble heritage we received.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1950 GENERAL PRACTITIONER AWARD

The Executive Committee of the State Society, after soliciting recommendations from all component societies and considering all data received, elected Dr. William A. Gillaspie, of New Orleans, as outstanding general practitioner of Louisiana for 1950. It is felt that this is an honor well deserved by Dr. Gillaspie and recommendation has been made to the Board of Trustees of the American Medical Association that consideration be given to selection of him for national recognition as outstanding general practitioner for this year. Below is biographical sketch of this member who should be commended for his achievements in medical practice during the past 54 years.

WILLIAM A. GILLASPIE, M. D.

Dr. William A. Gillaspie, Sr. this year completed 50 years of general practice in New Orleans. Looking back on his full and interesting life (he is now 77) it is difficult for him to explain exactly when and why he decided to study medicine.

Maybe, he says, the urge first appeared when he enthusiastically prepared mud pills for his playmates as a child. He also remembers that after a day of planting, binding or threshing on his father's farm in Columbia, Missouri, he was so tired he would say to himself, "There must be an easier way to make a living."

But the story of his successful life indicates that thoughts of his medical career must have been foremost with him always. He has practiced a total of 54 years and during his 50 years in New Orleans has served on the board of health many years, held numerous key assignments in public organizations, built up a large city practice and prepared two of his children to carry on his good work.

Dr. Gillaspie was born July 10, 1873, the first in a family of six boys. It was during his early education at the country school house and later at the Columbia high school that he helped maintain the family's 160-acre cattle, mule and horse farm.

In his junior year at the high school he took an entrance exam for the University of Missouri, also located at Columbia, passed and began studying for a Bachelor of Science degree. In 1893, two years after he entered college, his long felt desires for the study of medicine materialized.

Having enough credits to enter the medical school at the University, he enrolled as one of a total of three students.

Dr. Gillaspie flatly states that there is no comparison between the thorough medical schools of today and the one at which he studied before the turn of the century. Education then, he explains, was mainly one of theory. There was no hospital or major clinic attached to the University of Missouri, in fact the closest hospital in the area was at St. Louis, some 100 miles from Columbia.

One thing was in his favor. Because of the small enrollment he was invited to join the dean of the school, Dr. A. W. Mc Alister, on his rounds of medical practice in the surrounding counties. This first-hand observation helped him to knit the theory of his books with the practical tactics of doctoring in the country. Conditions naturally were primitive, miles deep into the woods, but what Dr. Gillaspie missed of modern methods of medicine of that day, he gained in what he calls "watching nature cure the sick" when other attention was too late or helpless under the circumstances.

Then came graduation in 1896 and the right to practice for himself. Interning then was not required.

Today Dr. Gillaspie, after living in New Orleans so long, often refers to a city in Missouri where

he was once located as "a small town of about 3,000", but the place where he got his start was by far smaller.

His father had for years been suffering with a chronic stomach ailment and his condition was on the decline when Dr. Gillaspie was graduated in 1896. To be close at hand he went about 10 miles north of Columbia to a little town called Brown's Station to begin his way in the medical world. The station at that time had a population of approximately 150 and oddly enough was named after the Dr. Brown whom he went to assist. Old Dr. Brown had been consultant for four or five of the neighboring counties for many years and it was not an easy process for the "young upstart" to convince the people that he was anywhere near as efficient as the man for whom he worked.

Dr. Gillaspie clearly remembers the case that labeled him "Doc" with the people of Brown's Station. He laughs a little when he tells the story today.

It was one of those days when no one knew where Dr. Brown was. Four or five men rushed into office and told young Dr. Gillaspie that he would have to ride with them and attend a young girl who was having convulsions.

The doctor saddled up his mule, threw on the saddle bags and followed the men some seven miles into the country. When he walked into the shack he was appalled to see about 10 women trying to hold the young girl down. He quickly tried her pulse and made the decision that the girl was merely suffering from hysteria.

"Leave the girl alone," he told the women.

"Oh, doctor, she'll hurt herself," they wailed.

"If she does, she'll calm down," he told them sternly.

So while the girl continued to throw herself around the bed he went over to his bags in the corner of the room and tried to decide just what to give her. Finally the solution hit him like a flash. If he gave the girl something to really be sick about he figured she would forget her hysteria.

"Look Millie," he told her, "I'm going to give this to you and it's going to cure you. You're going to be the sickest gal around these parts for a while but you won't be troubled with convulsions anymore."

With the preliminaries over, he administered one-tenth of a grain of apomorphine and then Millie *was* sick. After her seige she fell off to sleep, woke up feeling fine and as far as Dr. Gillaspie heard was never troubled again with convulsions.

All total it was a 14-mile trip through a driving sleet and he never got paid for the call, but at least it made the people consider him as a doctor. He calculated the time was well spent.

Looking back on his days at Brown's Station Dr. Gillaspie remembers most keenly the way he and his co-workers had to be pharmacists as well as doctors. The few drug stores had medicines in bulk but the task of filling prescriptions and making pills fell to them. Good experience, for sure, but time-consuming also.

After seven months at the station Dr. Gillaspie was called back to his home in Columbia to attend the final hours of his father. The man died in his son's arms.

With his father gone there was no reason to return to the little station so he struck north to Madison, Missouri, at the request of the town's banker, a former schoolmate of his. Madison's Doctor Hewland, was ready to retire so he took over the practice.

Conditions in Madison, population about 3,000, were better than those at the station. Good roads enabled Dr. Gillaspie to change his conveyance from mule to horse and buggy when visiting his patients in the surrounding territory. He recalls his standard price of $2.00 for a home call and $1.00 for an office visit—when he collected. Just like the other country doctors of his day he often took his payment in apples, peaches, potatoes or just plain friendship.

While practicing at Madison he married the cousin of his banker friend, Miss Sloan Atterbury. Her father, also a doctor, had moved south to Estelle, Miss., where after finding his practice slow, he began raising cotton. Soon he had increased the size of his farm to 5000 acres with a hundred or more colored laborers living on the grounds to work the fields.

This progressive little cotton farm was to play a large part in the life of Dr. Gillaspie. During a Christmas visit with his in-laws he was persuaded to leave Madison and join the family to care for the plantation's help.

It was pleasant living on the plush southern farm far removed from the below zero weather of his native Missouri. But tragedy struck into his life. His young wife was willed by a train while strolling along the tracks near the home.

After the accident Dr. Gillaspie knew that he needed a change of environment and having heard of the prominence of Tulane University's medical school he decided to come to New Orleans and advance his education.

Missouri's medical dean gave him good recommendations and by successfully taking junior and senior examinations he earned his degree in 1900 after one year of study.

His country life was behind him. Now he realized that he could never leave New Orleans. To him, it represented the very hub of the country and because of its busy port connecting it with other parts of the world, a doctor could find every imaginable disease within its limits. During his stay at Tulane he had seen and treated cases of

leprosy, bubonic plague and yellow fever, to mention a few.

But upon his second graduation a new problem confronted him. In the country, practice was slow, but assured. Here in the city the competition was keen and young doctors had to plan their careers carefully or be lost in the shuffle.

There was one practical move, to become a physician for an organization that offered its members medical treatment.

Naturally the field was somewhat crowded. This is where an undiscovered trait in Dr. Gillaspie's personality found expression, a trait that had little chance to develop in country practice. He found himself with an ability to hold his own in the stepped-up race for success in a large city.

Finding no suitable organization with which to collaborate he simply decided to form his own. Calling it the "Chicago Star" he and a few of his relatives formed the company which gave free medical care and medicines to its members. The Star grew rapidly in membership and Dr. Gillaspie found himself with patients to spare but with only a small financial gain. The groundwork was valuable. When six months later he sold his organization to a larger concern because of its growing size he quickly found that his patients continued coming to him although the new owners offered free medical service by another doctor.

As with every medical man who practiced in New Orleans at the turn of the century, Dr. Gillaspie played his part in the severe yellow fever epidemic which hit the city. He had 30 cases of his own and came down with the dreaded sickness himself.

In the similar flu epidemic of 1918 he made as many as 100 visits a day while working from 6 in the morning until midnight.

He recalls the familiar procedure of the family being treated to ask that he stop next door before leaving.

"Don't ring the bell," they would say, "just go right in, they are all sick in bed."

He ate on the run and never saw home except for the few hours sleep each night which he guarded carefully to restore his energy for the following day.

After beginning his new life in the city, Dr. Gillaspie married the former Helen Byrne of New Orleans in 1903.

Shortly after his graduation from Tulane he hung his shingle under that of Dr. Sara Napp, a close friend, in a small office at Canal and Rampart Streets.

A few weeks later he was notified that he was delinquent on his professional tax. Dr. Gillaspie contacted a rather important politician at his Elks club and explained that he hadn't ben in business long enough to run up any kind of tax.

The political figure shrugged the young doctor away by saying, "I can't help you, I don't even know you."

"Maybe you don't now," Dr. Gillaspie said, "but one day you will."

Later the same year the politician offered Dr. Gillaspie a vacant position on the city board of health which he held for four years.

Then things began to happen fast to this country doctor whom circumstances had sent to New Orleans. He was appointed inspector for the board of health and held the post for many years.

His practice began growing and in 1904 he moved his office into the Macheca building now occupied by Godchaux's clothing store.

Within the next 10 years he was appointed team physician for the New Orleans Baseball Association, medical examiner for the Louisiana State Boxing Commission and physician for the old Louisiana Jockey Club.

Naturally he was kept quite busy with these duties added to his regular practice. But the time element coincided with his schedule well. He would be at his office until 3 in the afternoon, at the race track until 3:30 and at the ball park for the rest of the day. In between time he was busy examining fighters before their appearances in the ring. To mention a few that passed before him: Max Schmelling, Gene Tunney, Primo Carnero and Jack Dempsey could be listed.

Dr. Gillaspie was holding some of these extra assignments for most of his fifty years in New Orleans. His longest term was with the baseball association, a job he relinquished just a few years ago. This, he says, was his favorite.

"I would have been at the ball park in my spare time anyway. That's my dessert."

But during the 50-year period he also found time to serve as physician for the New Orleans Police Department for 14 years.

Now that he has turned the half-century mark, Dr. Gillaspie is beginning to slow down his pace. One position he still holds is staff member at Hotel Dieu where he has practiced since his graduation in 1900.

Far from being the end of the story of Gillaspie the doctor, it would be more appropriate to call 1950 the beginning. In his office at the Audubon Building, where he is now one of the oldest tenants, you can't ask for Dr. Gillaspie without getting the receptionist confused. You have to pick your choice, one of three.

Together with Dr. W. A. Gillaspie, Sr., there is Dr. J. P. Gillaspie, eye, ear, nose and throat specialist, and Dr. R. E. Gillaspie, general practitioner, surgeon and also assistant coroner for the city of New Orleans. Both are his sons.

The oldest member of the firm very plainly cannot look into the future without seeing his sons. He explains that they both spent the full four years at college getting their Bachelor's degrees

to make sure they wanted to study medicine. They were not to be persuaded.

Besides the two young doctors there are two other sons, W. A. Jr., now a practicing attorney in Thomasville, Ala., and Charles Arion, who works with the Major of the traffic department on the New Orleans police force.

Of the three daughters, one is a housewife, another a specialized teacher with the New Orleans school board and the other—a medical technician in the office of Gillaspie, Gillaspie and Gillaspie.

Their father explains that the children had as much education as they wanted, simply because that was his investment. To him it was a sure thing.

He has set a goal for them in their own profession. In 1946 Dr. Gillaspie was honored by the University of Missouri for his 50 years of medical work. This year he was similarly honored by Tulane University and the Louisiana State Medical Society at Baton Rouge.

Looking back on his own career the doctor reflects that in his opinion a tour of country duty is a valuable thing for a young man getting his start in medicine.

"Of course being a country doctor nowadays is easier than it was 50 years ago," he says, "but it's still harder to take than the advantages of a big city. It's the experience that evens up the scale."

But he also adds that no matter where you practice your studies are never over. He has kept up with the advancements of his profession through associations at medical meetings, through his connections at Hotel Dieu and of course by keeping abreast of his two doctor sons.

His medical meetings are furnished by his membership in the Orleans Parish Medical Society, the Louisiana State Medical Society, the American Medical Association, and the American Academy of General Practice.

Other than medical, his memberships are almost too numerous to mention. "Doctors are joiners," he says, "they have to meet and know people."

Two organizations with which he is still quite active are the Elks and his Masonic Order in which he is a Shriner.

Now that he has reached the age of 77 and has two young sons to carry the load of his practice he is planning to slow down a little.

"Don't you think it's about time?" he asks.

"I believe that when a man gets to be my age he should have an interesting hobby to absorb his interest and his energy to keep him from going stale."

And when asked what hobby was to fill his later years he quickly answers, "Why my profession, of course."

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

FOURTEENTH ANNUAL MEETING OF THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The fourteenth annual meeting of The New Orleans Graduate Medical Assembly will be held March 5-8, headquarters at the Municipal Auditorium.

Nineteen outstanding guest speakers will participate and their presentations will be of interest to both specialists and general practitioners. The program will include a panel discussion of ACTH and Cortisone, a series of talks on trauma and neoplastic disease, a review of the application of radioactive isotopes in medical practice, clinicopathologic conferences, round-table luncheon discussions and many other features of special interest.

Another attraction of the meeting will be daily demonstrations of medical and surgical procedures in color television. This program will be telecast from Charity Hospital to the auditorium and is sponsored by Smith, Kline & French Laboratories.

The Assembly has planned another interesting postclinical tour to follow the 1951 meeting in New Orleans. On Saturday, March 10, a party composed of doctors and their families will leave by plane for Panama. The itinerary also includes Medellin and Cali, Colombia; Quito, the capital of Ecuador and Lima, Peru. Medical programs and visits to hospitals have been arranged, together with a full schedule of sightseeing. The group will return to New Orleans on Sunday, March 25. Details and a complete itinerary are available at the office of the Assembly, Room 103, 1430 Tulane Avenue, New Orleans 12, La.

AMERICAN UROLOGICAL SOCIETY SEMINAR

The South Central Branch of the American Urological Society will hold its Seminar in Dallas at the Adolphus Hotel, January 29 through February 2.

PLANS FOR CRIPPLED CHILDREN'S HOSPITAL

A campaign to complete the building fund for a 50 bed convalescent hospital for crippled children will begin this month. The Crippled Children's Hospital wil loccupy a site at the corner of Henry Clay and Tchoupitoulas Streets adjacent to Audubon Park in New Orleans. It will serve children from the entire state.

The cost of the Crippled Children's Hospital building and stationary equipment is set at $659,350. This, and other costs such as improving the site, landscaping, furnishing, etc., will be met by $500,000 from public subscription and by a Louisiana State Hospital Board allocation of $315,000 in Federal funds to the project.

The hospital will be a one-story building of contemporary design. Open planning, patios and large window walls will distinguish it from the usual institutional building.

The hospital's outpatient department will be equipped with its own examining rooms, plaster room, bracemaker's room and lecture auditorium. The department will share physical therapy, diathermy, infra red and ultra violet treatment rooms, X-ray and laboratory facilities with the hospital proper.

Inpatients will be housed in 6-bed units with isolation rooms available for problem cases. A "homelike" atmosphere will be maintained by grouping all patient living areas with the bedrooms, separate from the treatment areas, and by the use of color instead of the traditional white. The emphasis on rehabilitation is evident in the inclusion of school rooms, a music room, a library and a large occupational therapy department.

The hospital will have an open staff. It will be a non-sectarian, non-discriminatory, non-profit institution, accepting convalescent cases from birth to adulthood, with the concentration of patients in the seven-to-fifteen age range. Private patients will be admitted, as well as those referred by the Section of Crippled Children's Services of the State Department of Health and the National Foundation for Infantile Paralysis, and other qualified agencies.

THE LOUISIANA HEART ASSOCIATION WELCOMES REQUESTS FOR GRANTS IN AID

The Louisiana Heart Association announces that it will welcome requests for grants in aid for research in cardiovascular disease and in related fields. The deadline for receipt of requests is named as January 2, 1951.

Application blanks should be obtained from the office of the Louisiana Heart Association and requests addressed to Dr. Russell Holman, Chairman, Research & Fellowship Committee, Louisiana Heart Association, Inc., Room 103, 1430 Tulane Ave., New Orleans, La.

Applications received by January 2 will be reviewed on or about January 10, 1951. It is anticipated that funds will be available for research allocations on July 1, 1951, out of the Association's 1951 Heart Campaign receipts. Preference will be given to requests from Louisiana.

AMERICAN COLLEGE OF SURGEONS MEETS IN ST. LOUIS

A cordial invitation has been extended to the medical profession in New Orleans to attend a three-day Sectional Meeting of the American College of Surgeons in St. Louis on January 22, 23 and 24. The Statler Hotel will be headquarters for the meeting and requests for hotel accommodations should be directed to the Statler Hotel in St. Louis.

The program for this meeting will include new surgical motion pictures, a special program on trauma, a cancer symposium, and panels or papers on Vascular Surgery, Chest Injuries, Fractures about the Ankle Joint, Hematuria following Trauma, Neck Surgery, Osteomyelitis, Ulcerative Colitis, Cancer of the Stomach, and Emergencies Arising During Operation. The first two days of the program will be presented at the headquarters hotel, and on January 24, the hospitals in St. Louis will offer a full day of surgical clinics for those in attendance at the meeting.

A five-dollar registration fee will be required except from Fellows and members of the Junior and Senior Candidate Groups of the College, and internes and residents, but the physician or surgeon in practice will find the program worth many times the registration fee.

RELATIONSHIP BETWEEN ADRENAL STEROIDS AND PSYCHOSIS

Clues to the role in psychosis of ACTH, cortisone and similar hormones will be studied in a cooperative project by the National Institute of Mental Health of the Public Health Service, and the Worcester Foundation for Experimental Biology, Shrewsbury, Massachusetts, Dr. Leonard A. Scheele, Surgeon General of the Public Health Service, announced recently.

Recent evidence points to a relation between these hormones and mental illness. The new investigations at the Worcester Foundation will be primarily concerned with the study of differences between the output of adrenal cortical hormones in schizophrenic patients and in well persons. New techniques of hormone analysis used by the Foun-

dation promise to reveal facts about specific chemical mechanisms that may help explain certain types of mental illness. From such basic studies, it is hoped, new methods of treatment may eventually be developed.

The joint project will be under the direction of Dr. Hudson Hoagland, Executive Director of the Worcester Foundation and consultant to the Public Health Service's National Institute of Mental Health.

WOMAN'S AUXILIARY OF THE ORLEANS PARISH MEDICAL SOCIETY

The Orleans Parish Auxiliary was very proud to have the honor of a visit from our National President, Mrs. A. A. Herold, and our State President, Mrs. DeWitt T. Milam on the same day. A luncheon was given in honor of these two very distinguished Auxiliary leaders—our "Big Mamma" as we in New Orleans think of Mrs. Herold, inasmuch as it was through her and Dr. Herold's efforts the State Auxiliary was organized at a Convention held in New Orleans in 1929, and our "Little" Mamma of the State Auxiliary, who has given all of us much inspiration and encouragement as only Mrs. Milam could do. Mrs. Herold stressed the importance of our participation in the Nurse Recruitment Program and Civil Defense, and A.M.A.'s 12-point program; she also urged a program of self-education among Auxiliary members to be able to intelligently promote the foregoing projects. Mrs. Milam requested assistance of the members in enlarging our Auxiliary membership to match that of the doctors. She too, stressed the importance of Nurse Recruitment, enrollment in Blue Cross and Blue Shield and Periodic Health Examinations. Participation in all civic and community projects, particularly of a health nature, is a must for every doctor's wife.

Members of the Orleans Parish Auxiliary were indeed very much enlightened as to the national and state programs and were very happy to have had this information given them first hand by the charming presidents of the two auxiliaries they represent.

MRS. GEORGE D. FELDNER,
Publicity Chairman.

WOMAN'S AUXILIARY TO THE LOUISIANA ACADEMY OF GENERAL PRACTICE

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Eighth District—Mrs. N. M. Brian, Jr., 2333 Hill St., Alexandria, La.

MRS. J. OTIS BROYLES,
Publicity Chairman.

BOOK REVIEWS

Century of Medicine in Jacksonville and Duval County: By Webster Merritt, M. D., Gainesville, Fla. Univ. of Florida Pr. 1949, Illus, 201p. Price, $3.00.

Newest of the County medical histories is this attractive and unusually interesting one of the Jacksonville, Florida area, which covers the period of the eighteenth century. Settlement here was much later than on the West coast of Florida, the first settler having arrived in 1791.

The story as related contains information as to the earliest physicians, epidemics of yellow fever, small pox, cholera, dengue, and malaria, health conditions and the problems of sanitation, with activity toward their solution and the founding of the State Board of Health. An account of early effort toward medical licensure is included as well as histories of early hospitals and an attempt toward establishment of a Medical School. Many of the problems as described are those which faced the medical pioneers in our own area, which lends to the book a special interest for us. Medical developments are well correlated with the happenings of the general history and with the history of medicine in the United States.

The book is profusely illustrated with portraits of physicians, as well as sketches and photographs of contemporary views. It is well documented throughout with specific bibliographic references to source material. Locally colored with quotations of letters and reminiscent interviews with many of Florida's older physicians, this volume represents a real reference book for the period and locality portrayed.

MARY LOUISE MARSHALL.

Your Nasal Sinuses and Their Disorders: By Albert P. Seltzer, M. D., M. Sc., Sc. D (Med.), F. A. C. S., F. I. C. S. Froben Press, 1949. Illus. 155p. Price, $2.50.

This book consists of one hundred and fifty-two pages, divided into sixteen chapters, with twenty-four illustrations. It is not prescribed as a text book but it should serve the laity as a source of preventive medicine.

It is well written by one who has plenty of clinical experience and who warns against patients making their own diagnosis of sinus trouble, and using treatments which harm the delicate membranes of the nose and sinuses.

The illustrations are splendid for the otolaryngologist or any physician to have in his office to demonstrate the location of the sinuses to the patient.

It presents no new facts to the specialist but it does translate them into simple language that we can convey to our patients.

MONTE F. MEYER, M. D.

A Textbook of Neuropathology, with Clinical, Anatomical and Technical Supplements: By Ben W. Lichtenstein, B.S., M.S., M.D. Saunders, 1949. Illus. pp. 474. Price, $9.50.

This is a well organized and nicely illustrated textbook. In reading it one can immediately gather that the author has had at his disposal a rich and excellent collection of pathological material and that, undoubtedly, he has made good use of it. The chapter on neoplasms appears the best. The neuroanatomical and the neuropathological technic supplements, as well as the listing of syndromes and uncommon diseases, are useful additions. References are extensively covered.

As the author points out, this textbook is primarily for the medical student and for those training in neurology, neurological surgery, pathology, and psychiatry.

FRANCISCO GARCIA-BENGOCHEA, M. D.

The Nose—An Experimental Study of Reactions within the Nose in Human Subjects During Varying Life Experiences: By Drs. T. H. Holmes, Helen Goodell, Stewart Wolf, and Harold G. Wolff.

This is a very interesting book and although the authors have spent a lot of time on these experiments, many must still be put to practical use. I wish they would have gone further in detail in these cases of pregnancy where they have a blocked nose throughout pregnancy, even in prospective mothers who want children. We have thought this blockage was due to an allergy of the products of pregnancy and not because they were unhappy or frustrated individuals.

With the limitations and encroachments of the otolaryngological field by the antibiotics and the antihistaminics, maybe this will be our salvation as we will all become otolaryngological psychiatrists.

MONTE F. MEYER, M. D.

Some Relations between Vision and Audition: By J. Donald Harris, Ph.D. Charles C. Thomas, Publisher, 1950. pp. 56. Price, $1.50.

This well documented monograph analyzes the similarities and dissimilarities between the physical aspects of light and sound perception. Sensitivity, effect of intensity range, perceptual growth and decay, perceptual acuity differences for stimuli of varying wave lengths, and visual and auditory flicker perception are evaluated. Intersensory facilitation is examined in the light of present-day anatomic, physiologic, and psychologic knowledge. It is concluded that the theoretical possibility of intersensory enhancement is undebatable. The material, although of a somewhat technical nature is

quite readable and should serve as a stimulus to further thought and investigation of the subject.

GUSTAV C. BAHN, M. D.

Proctology in General Practice: By J. Peerman Nesselrod, B.S., M.S., M.Sc. (Med.), M.D., F.A.C.S., F.A.P.S., Philadelphia, W. B. Saunders Co., 1950. 276 pp. Price, $6.00.

The author has contributed a well written and interesting book that contains much of great practical value to all physicians in general and to internists, proctologists, and general surgeons in particular. He gives the reader the essential and pertinent facts in each of the proctologic problems presented without going into unnecessary details. Of particular and very prectical value are the chapters on diagnostic procedures and clinical proctoscopy. In each of these the lessons learned in an extensive clinical experience are passed on to the reader.

FRANK L. LORIA, M. D.

Post-Graduate Lectures on Orthopedic Diagnosis and Indications, Vol. I: By Arthur Steindler, M.D., F.A.C.S., Springfield, Ill., Charles C. Thomas, Publisher, 1950. pp. 302, 400 illus. Price, $7.50.

This monograph is the first volume of a contemplated four volume set presented in the same lecture form as used by Dr. Steindler in graduate teaching at the University of Iowa for the past thirty years. As the author points out in the preface, these lectures are not intended as a text or description of purely technical procedures, but are intended to cover diagnosis and indications.

The book is divided into two sections, the first covering propedeutics, or the preliminaries to the study of orthopedics, which are covered in a set of four lectures, the first being symmetry and asymmetry of the body; the second, contractures, the third, interpretation of pain in orthopedic surgery; and fourth, the pathology of gait. The second section of the book is on congenital deformities and disabilities which are presented in a form of seven lectures. The first is general observations on congenital deformities of the locomotor system; the second on congenital deformities due to disturbance of bone growth; the third is on congenital deformities of the spine and thorax; the fourth on congenital calipes; the fifth on defects and malformations of the long bones of the lower extremity; the sixth on congenital dislocations of the hip; and the seventh on congenital deformities of the upper extremity.

The first section of this book is not read with ease or without dilligent study of the material presented. The author in his attempt to obtain accuracy in alignment, joint motion, and gait frequently obscures the point which he is attempting to make, as much as the title of his first section, propedeutics, does. His discussion of interpretation of pain in orthopedic surgery is excellent.

The second section of his book presents a great number of clinical entities representing the various congenital deformities of the musculoskeletal system. Its greatest value is that it represents a very comprehensive and complete compilation and classification of the various congenital deformities. It is more comprehensive and complete than any this reviewer is familiar with. It is illustrated profusely with photographs and roentgenograms from the author's personal collection.

As a whole, this condensed series of lectures is well presented, profusely illustrated, with extensive bibliographies on each chapter. This first volume on postgraduate lectures will be extremely helpful to students in orthopedic surgery. The reviewer doubts whether those not intimately concerned with orthopedic surgery will be willing to take the time to digest the material that is presented in this book.

JACK WICKSTROM, M. D.

PUBLICATIONS RECEIVED

Grune & Stratton, New York: Differential Diagnosis of Internal Diseases, by Julius Bauer, M. D., F. A. C. P.

Oxford University Press, Inc., New York: Supplements to Oxford Loose-Leaf Medicine.

Philosophical Library, New York: Principles of General Psychopathology, by Siegfried Fischer, M. D.

W. B. Saunders Company, Philadelphia: A Text-Book of X-Ray Diagnosis, by British authors in four volumes, edited by S. Cochrane Shanks, M. D., and Peter Kerley, M. D. (2nd Edition); An Integrated Practice of Medicine (Progress Volume), by Harold Thomas Hyman, M. D.; Researches in Binocular Vision, by Kenneth N. Ogle, Ph. D.; Regional Orthopedic Surgery, by Paul C. Colonna, M. D.; Bronchoesophagology, by Chevalier Jackson, M. D., Sc. D., LL. D., F. A. C. S., and Chevalier L. Jackson, M. D., M. Sc., F. A. C. S.

Charles C. Thomas, Publisher, Springfield, Ill.: Radiation Therapy in the Management of Cancer of the Uterine Cervix, by Simeon T. Cantril, M. D.; Urgent Diagnosis Without Laboratory Aid, by Prof. Dr. Hanns L. Baur; Thromboembolic Conditions and Their Treatment with Anticoagulants, by Charles D. Marple, M. D., and Irving S. Wright, M. D.; Newer Concepts of Inflammation, by Valy Menkin, M. A., M. D.; Basic Principles of Clinical Electrocardiography, by Hans H. Hecht, M. D.